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DRAKE CHEMICAL INCINERATOR RISK ASSESSMENT

Prepared for

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EXECUTIVE SUMMARY

The Drake Chemical Site in Lock Haven, Pennsylvania is an inactive chemical manufacturing facility that operated from the early 1940's through 1982. Dye chemicals and dye chemical intermediates were manufactured on the site for more than three decades. During this time, there were unlined lagoons containing pretreatment sludge, demolition debris, chemical tanks and reactors, and several thousand rusted and leaking drums containing various chemicals.

Since 1982, the U.S. Environmental Protection Agency (EPA) has been involved in ongoing remediation activities at the site. The current Remedial Action involves excavation of approximately 200,000 cubic yards of contaminated soil that will be treated by a rotary kiln incinerator constructed on the site. The trial burn for the incinerator is scheduled for January 1996, with full operation by the spring of 1996. The incinerator is scheduled to be in operation for approximately two years.

The EPA Regional Air Offices are routinely involved in the analysis and evaluation of air impacts from Remedial Actions at Superfund sites. This risk assessment evaluated the potential health impacts to nearby residents during the two-month period of the proposed trial burn only. Health risks from inhalation exposure to estimated emissions from the incinerator were assessed for both children and adults to determine if the trial burn period itself represented a potential adverse impact to local residents.

The risk assessment process involved a number of steps including the estimation of emissions, the dispersion modeling analysis, the exposure assessment, toxicity assessment, and concluding with the risk characterization process. An uncertainty analysis which quantitatively describes some of the uncertainty and conservatism built into the analysis was also included.

The emissions were calculated based on soil concentration data and estimated pollution control efficiencies. Off-site air concentrations were developed based on the emission rates

and U.S. EPA approved dispersion modeling. The evaluation of the impacts of the estimated chemical air concentrations on hypothetically exposed individuals in the surrounding area was accomplished by comparing predicted maximum concentrations to applicable toxicological criteria.

The results of the analysis indicated that no adverse health impacts are anticipated due to the emissions from the incinerator during the trial burn for either children or adults. This analysis evaluated carcinogenic and noncarcinogenic risks, compared predicted criteria pollutant concentrations to Natural Ambient Air Quality Standards (NAAQS), and evaluated potential blood lead levels in children. The predicted carcinogenic risks were less than one chance in a million for both children and adults, which is below the low end of the U.S. EPA acceptable risk range. The predicted noncarcinogenic risks were all less than a hazard index of one, and the criteria pollutant and lead analyses indicated that these pollutants were not of concern from a health perspective. It was concluded, based on these analyses, that the emissions from the incinerator during the two-month period of the trial burn will not cause any adverse health impacts to nearby residents.

SECTION 1

INTRODUCTION

1.1 BACKGROUND

The Drake Chemical Site in Lock Haven, Pennsylvania is an inactive chemical manufacturing facility that operated from the early 1940's through 1982. The site is located in Lock Haven directly across Bald Eagle Creek from the town of Castanea. Dye chemicals and dye chemical intermediates were manufactured on the site for more than three decades. During this time, there were unlined lagoons containing pretreatment sludge, demolition debris, chemical tanks and reactors, and several thousand rusted and leaking drums containing various chemicals.

Since 1982, the U.S. Environmental Protection Agency (EPA) has been involved in ongoing remediation activities at the site. Although all process wastes and buildings were removed during these past activities, previous studies conducted on the site indicated that the remaining contamination may still present a serious threat to the environment and human health. The current Remedial Action involves excavation of approximately 200,000 cubic yards of contaminated soil over the 9 acre site to a depth of 12 feet below the surface. The contaminated soil and sludge mixture will be treated by a rotary kiln incinerator which will be constructed on the site. The trial burn for the incinerator is scheduled for January 1996, with full operation by the spring of 1996. Figure 1-1 is a map showing the location of the incinerator in Lock Haven.

1.2 OBJECTIVES

The EPA Regional Air Offices are routinely involved in the analysis and evaluation of air impacts from Remedial Actions at Superfund sites. This risk assessment evaluates the potential health impacts to nearby residents of on-site high temperature incineration. The analysis focused on the two-month period of the proposed trial burn only. Health risks from exposure to estimated emissions from the incinerator were assessed for both hypothetical
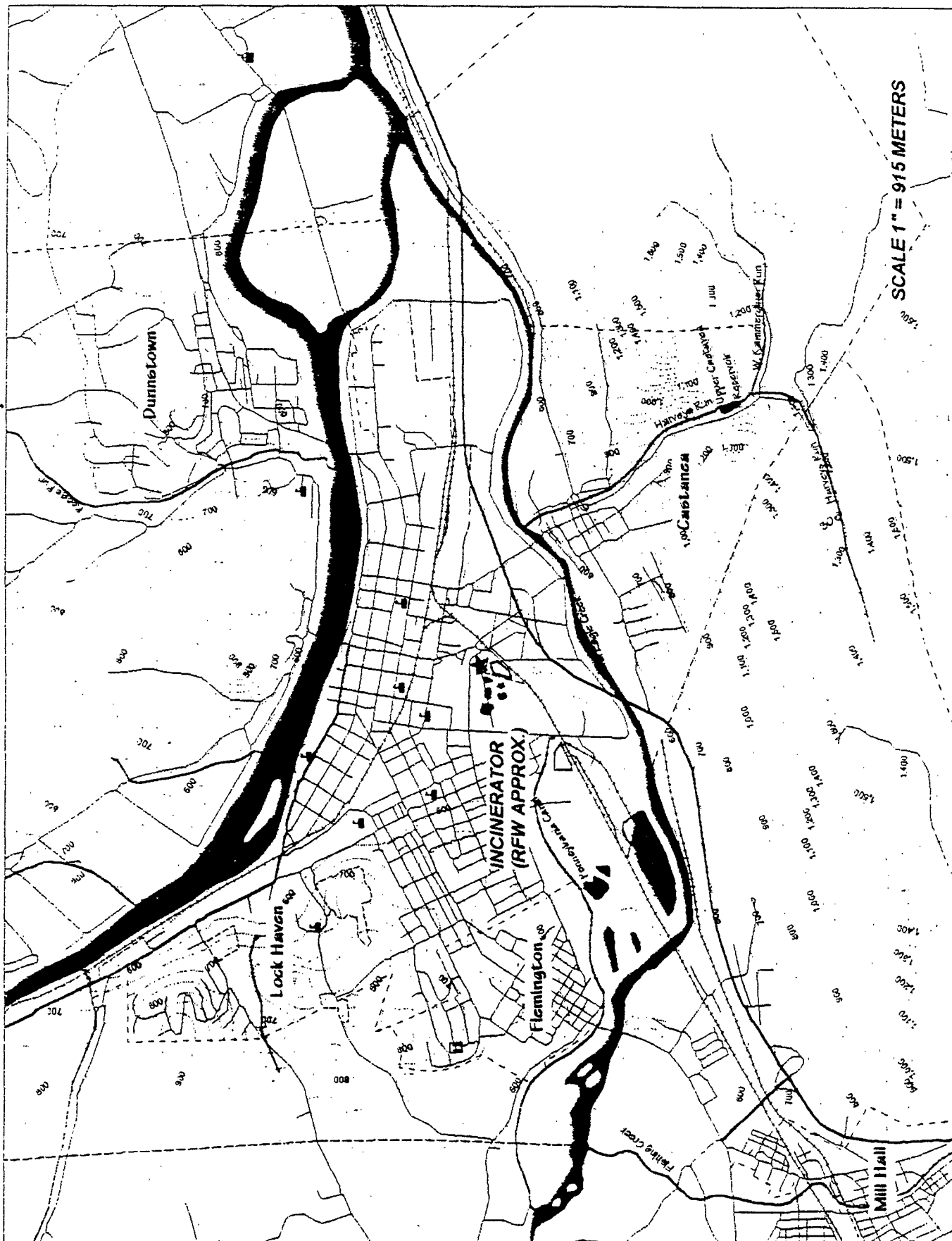
FIGURE 1-1
LOCATION OF INCINERATOR

SCALE 1" = 915 METERS

child and adult receptors to determine if the trial burn period itself represented a potential adverse impact to the local residents.

1.3 APPROACH

The emissions were calculated based on soil concentration data and estimated control efficiencies. Off-site air concentrations were developed based on the emission rates and dispersion modeling. The evaluation of the impacts of the estimated air concentrations on hypothetically exposed individuals in the surrounding area was accomplished by comparing predicted concentrations to applicable toxicological criteria. This approach is consistent with evaluation of inhalation risks according to EPA (1989a). Figure 1-2 provides a conceptual flow chart of the risk assessment process.

Section 2 of this risk assessment contains a brief description of the incinerator system, including the air pollution control system and the stack emission parameters. Section 3 identifies the predicted pollutants, their estimated emission rates, and the processes by which these pollutants and emission rates were determined. Section 4 describes the dispersion modeling analysis and provides predicted air concentrations of the emitted contaminants at the peak off-site locations. Section 5 is the risk assessment. This section includes: the exposure assessment, which estimates potential exposure for the hypothetical receptors through inhalation; the toxicity assessment, which identifies applicable health criteria; and the risk characterization, which integrates the exposure and toxicity assessments to determine potential health risks. An uncertainty analysis, which evaluates the possible effects of the assumptions on the results of the risk assessment, is also included in Section 5. Section 6 provides the applicable conclusions and Section 7 includes the references from all the sections of the report. Appendix A contains brief toxicity profiles for the chemical of potenial concern for the Drake Chemical site.
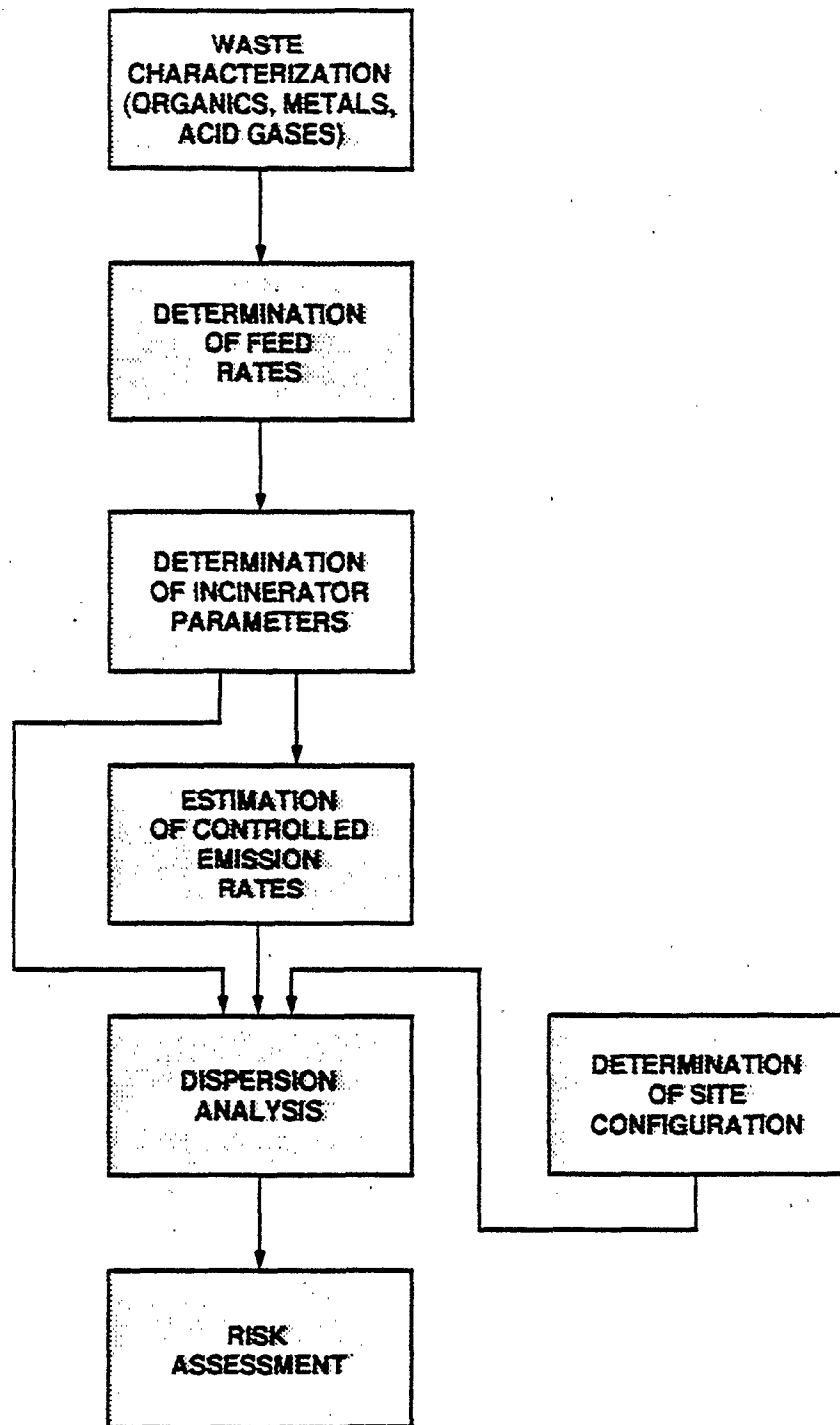
FIGURE 1-2   CONCEPTUAL FLOW CHART OF RISK ASSESSMENT
PROCESS FOR INCINERATION

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SECTION 2
DESCRIPTION OF THE INCINERATOR SYSTEM

2.1 GENERAL INFORMATION

RUST International, Inc. will use a transportable rotary kiln incinerator to treat the contaminated soils at the Drake Chemical site. The incinerator system includes a rotary kiln incinerator which allows for the processing of a wide range of soil types and consistencies. A more detailed discussion of the system is included in the trial burn plan.

.The incinerator system consists of the following components:

- Waste feed systems.
- Rotary kiln incinerator.
- Ash discharge systems.
- Cyclone and secondary combustion chambers (SCC).
- Air pollution control (APC) System.
- Utilities.

Figure 2-1 is a schematic of the incineration system. The contaminated soil will be fed to the kiln. Organic contaminants will be volatilized in the kiln and will exit with the hot gases into the secondary combustion chamber (SCC) where additional destruction of organics occurs. Cyclones ahead of the SCC will remove large particulate matter. A water quench will reduce the gas temperature, a baghouse will remove smaller particulate matter, and a scrubber will remove acid gases prior to release to the atmosphere from the stack. The specific stack parameters for the incinerator are discussed in Section 4.

TO
ATMOSPHERE

AIR POLLUTION CONTROL SYSTEM

| ACID GAS SCRUBBER | ← | BAG HOUSE | ← | QUENCH | ← |

INCINERATOR SYSTEM (60mmBTUs/hr)

STACK
HEIGHT - 150 FEET
TEMPERATURE - 180° F

ROTARY KILN
(1000° F) → CYCLONES → SECONDARY COMBUSTION CHAMBER (1800° F)

CONTAMINATED SOIL
(60 TONS
PER HOUR)

"CLEAN"
SOIL
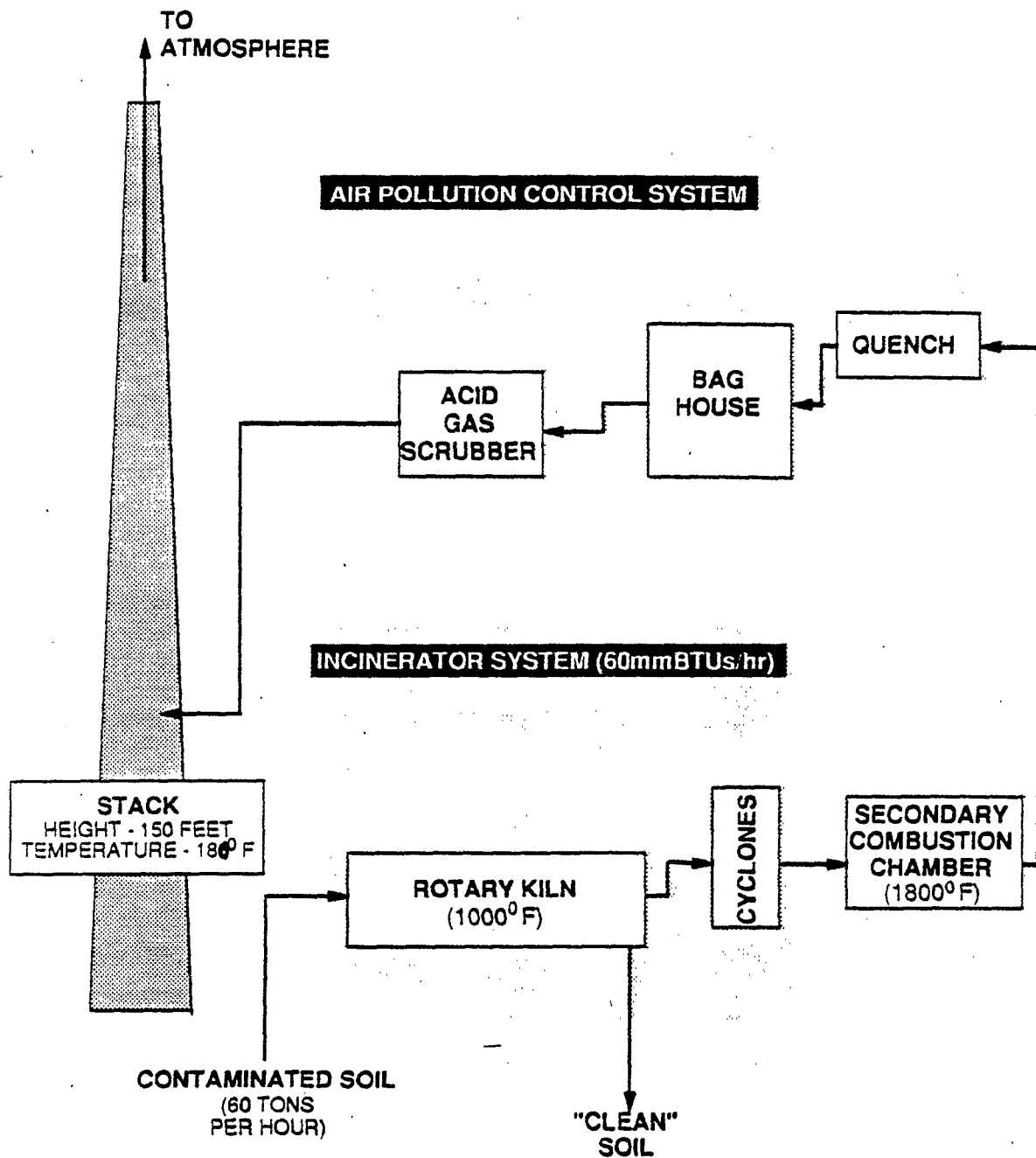
SOURCE: DRAKE CHEMICAL, 1995

DRAKE CHEMICAL SITE
LOCK HAVEN, PENNSYLVANIA

FIGURE 2-1
BLOCK DIAGRAM OF THE ROTARY KILN SYSTEM

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DRAKRA11-11/95G:\DM\HOME\C\RSKGRP

SECTION 3

DEVELOPMENT OF EMISSION RATES

3.1 APPROACH

A literature search was undertaken to obtain emissions and pollutant control efficiency data for similar incineration systems handling similar contaminants. Specifically, pollutant destruction and removal efficiencies for the combination of the rotary kiln incinerator and the proposed air pollution control devices were estimated. With this information, emissions from the incinerator system were predicted. The approach tended to overestimate rather than underestimate emissions, i.e., the emissions estimates are conservative.

3.2 AVAILABLE DATA

EPA provided a list of compounds to be considered in this risk assessment. EPA assessed the data characterizing the waste that will be burned in the rotary kiln and determined that the average and maximum concentrations for the organics and metals data reported in test pit soil analyses provided the best information (EPA, 1994a; 1994b). For those compounds not listed in the test pit analysis document, EPA directed that information be obtained from the October 1994 Test Pit Excavation Report prepared by Montgomery Watson (1994). Average and maximum emissions rates were established for each contaminant listed by EPA for which soil concentration data was available.

Pollutant control efficiency data were obtained from the U.S. EPA Office of Air Quality Planning and Standards (OAQPS) February 1992 report titled "Screening Procedures for Estimating the Air Impacts of Incineration at Superfund Sites" (EPA, 1992a). This guidance document provides an approach that has already been reviewed and found acceptable by EPA.

This document also provided organic destruction efficiencies in a rotary kiln incinerator/SCC unit, partition coefficients for metals (estimating the percentages that

remain behind in soil feed material or volatilize and are carried to the air pollution control devices), and the expected level of abatement for the given control equipment train. Further, an approach for estimating emissions of halogenated organic compounds (including guidance on a reasonable control efficiency for wet scrubbers) and a formula for estimating particulate matter emissions was provided (EPA, 1992a).

3.3 CALCULATION METHODOLOGY

The first step in the calculation procedure was to determine the total feed rate of each pollutant, i.e., volatile organic compounds (VOC), semi-volatile organic compounds (SVOC), metals, and chlorine, loaded to the incinerator system based on the average and maximum pollutant concentrations in the soil and the maximum incinerator capacity of 60 tons of soil per hour. The emissions were calculated using the properly selected factors from the EPA Screening Procedures (EPA, 1992a) and other technical guidance/data described in the following subsections.

3.3.1 Organic Compounds (VOC and SVOC)

The average and maximum feed rates for the organics were estimated using Equation 2 from the EPA Screening Procedures as follows (with a minor modification since the equation in the EPA document assumed concentrations in ppm, and the Drake Chemical analytical results are in ppb):

$$FR_0 = (FR)(C_0)(1 \times 10^{-9})$$

Where:

FR_0	=	Feed rate of organic compounds of interest, lb/hr
FR	=	Mass feed rate of waste to the incinerator, lb/hr
C_0	=	Concentration of the organic compound of interest, ppb
1×10^{-9}	=	Conversion factor for ppb

For the Drake Chemical incinerator, FR = 60 ton/hr = 120,000 lb/hr

Based on the EPA Screening Procedures on organic destruction efficiency in the rotary kiln incinerator and SCC, the principal organic hazardous constituents (POHCs), i.e, the volatile and semi-volatile compounds in the feed, can be assumed to be destroyed at 99.99 % efficiency. The applicable formula for emission estimations is equation (3):

$$ER_0 = (FR_0) \left[1 - \frac{DRE_0}{100} \right] (0.126)$$

Where:

ER_0 = The emission rate for each compound, g/s

FR_0 = Feed rate of each compound, lb/hr

DRE_0 = Destruction efficiency for the compounds, 99.99

0.126 = Conversion factor, lb/hr to g/sec

For the given incinerator soil feed rate and an estimated organic destruction efficiency of 99.99%, the two equations can be combined to produce the following:

$$ER_0 = (120,000) (C_0) (1 \times 10^{-9}) \left[1 - \frac{99.99}{100} \right] (0.126)$$

$$ER_0 = (1.512 \times 10^{-9}) (C_0)$$

3.3.2 Metals

The average and maximum feed rates for each metal are determined using Equation (4) from the EPA Screening Procedures:

$$FR_m = (FR)(C_m)(1 \times 10^{-6})$$

Where:

FR_m = Feed rate of metal species, lb/hr

FR = Feed rate of waste for the incinerator, lb/hr

C_m = Concentration of the metal species in the waste, ppm (or mg/kg)

1×10^{-6} = Conversion factor, dimensionless

The uncontrolled metal emission rate is calculated using Equation (5) from the EPA Screening Procedures:

$$ER_{mu} = (FR_m) \left[\frac{PF}{100} \right] (0.126)$$

Where:

ER_{mu} = Uncontrolled emission rate of metal species, lb/hr

FR_m = Feed rate of metal species, lb/hr

PF = Partition factor for metal species, %

0.126 = Conversion factor lb/hr to g/s

The partition factor is an estimate of the fraction of the feed of each metal that volatilizes and is ducted to the air pollution control devices. A limited listing of partition factors for metals is provided in Table 5 in the EPA Screening Procedures. For the selected metals, partition factors are given for two combustion gas temperatures, 1600°F and 2000°F.

The Drake Chemical incinerator operating temperature is expected to be approximately 1000°F. For this emission calculation, it was assumed the metals were partitioned the same as if the incinerator operated at 1600°F. This approach is conservative, i.e., the emissions will tend to be overestimated because more of the metal content will volatilize as the temperature is increased. Partition factors are provided for all metals of interest except nickel and selenium. Based on data provided in the Mitre Corp report, "Hazardous Waste Stream Trace Metal Concentrations and Emissions" U.S. EPA Office of Solid Waste, November, 1983 (MITRE, 1983), it was conservatively assumed that these two metals would be completely volatilized in the incinerator. Use of the partition data presented in the EPA Screening Procedures is also contingent upon the chlorine content of the waste feed stream being less than one weight percent. Calculations for acid gases (Section 3.3.3) indicate that

the chlorine concentration of the soil feed is less than one weight percent. Therefore, the EPA partition data is valid for this application. Further, for metal concentrations greater than 100 ppm in the incinerator material, as in the case of certain metals for the Drake Chemical incinerator feed stream, use of partitioning data in Table 5 could tend to overestimate metal volatilization. As such, use of the partition data in cases where incinerator feed metal concentrations exceeded 100 ppm would add another level of conservatism to the emission rate estimation for those metals.

The controlled emission rates of metals are estimated using Equation (6) from the EPA Screening Procedures:

$$ER_{mc} = (ER_{mu}) \left[1 - \frac{CE_m}{100} \right]$$

Where:

ER_{mc} = Controlled emission rate for the metal species, g/s

ER_{mu} = Uncontrolled emission rate for metal species, g/s

CE_m = Control efficiency for the air pollution control device (APCD).

Table 6 of the EPA Screening Procedures provides a listing of expected control efficiencies for various combinations of air pollution control devices for a limited selection of metals. The Drake Chemical incinerator APCD is a fabric filter followed by a wet acid gas scrubber (both preceded by a quench). This configuration is identified as FF/WS in the table. Again, estimated efficiencies are provided for the metals of interest in this analysis except nickel and selenium. The Mitre Corp. report (Mitre, 1983) provides additional data on expected metal removal efficiencies for various APCD. Based on the data provided in the Mitre report and in the EPA Screening Procedures, it was judged that the nickel APCD removal efficiency would be estimated to be the midpoint of the efficiencies given in the EPA document for arsenic (90%) and chromium (95%), since the Mitre study reported the removal efficiency of nickel by an electrostatic precipitator (ESP), which is another non-condensing particulate control device, in series with a wet scrubber to be between those for

arsenic and chromium. The ESP/wet scrubber removal efficiency reported in the Mitre study for selenium is greater than that for mercury and less than that for an antimony. Therefore, it is estimated that the APCD removal efficiency for selenium would be the midpoint of the removal efficiencies in the EPA guidance document for mercury (50%) and antimony (90%).

The equations for estimated metals emissions can be combined as follows using 120,000 lb/hr as the incinerator feed rate:

$$ER_{mc} = (120,000)(C_m)(1x10^{-6}) \left[\frac{PF}{100} \right] (0.126) \left[1 - \frac{CE_m}{100} \right]$$

$$ER_{mc} = (1.512 \times 10^{-6})(C_m)(PF)(100 - CE_m)$$

3.3.3 Acid Gases

From the chemical target list provided for this report, the only halogen capable of acid gas formation is chlorine which is a precursor to hydrogen chloride (HCl) formation.

The aggregate concentration of chlorine in the incinerator waste feed can be calculated Using Equation 7 of the EPA Screening Procedures:

$$C_A = \sum_{i=1}^{n} \left[(C_{Ai}) \left[\frac{MW_A}{MW_{Ai}} \right] \right]$$

Where:

C_A =   Concentration of acid-forming element in the waste, ppm

C_{Ai} =   Concentration of compound i containing the same element, ppm

MW_A =   Molecular weight of acid forming element, g/mole, times the number of the acid-forming elements in a molecule of compound i, N_{Ai}. (This is a clarification of the EPA Screening Procedures).

$$MW_{Ai} = \text{Molecular weight of compound i containing the same element, g/mole}_i.$$

The feed rate of each acid forming element is then calculated using Equation (8) of the EPA Screening Procedures:

$$FR_A = (FR)(C_A)(1x10^{-6})$$

Where:

FR_A = Feed rate of acid-forming element, lb/hr

FR = Mass feed rate of waste to the incinerator, lb/hr

C_A = Concentration of acid forming element, ppm

$1x10^{-6}$ = Conversion factor for ppm.

The uncontrolled emissions of acid gas can be calculated using Equation (9) from the EPA Screening Procedures:

$$ER_{Au} = (FR_A)(R_A)(0.126)$$

Where:

ER_{Au} = Uncontrolled emission rate of acid gas, g/s

FR_A = Feed rate of element, lb/hr

R_A = Stoichiometric ratio of acid gas-to-element, g/g (Table 7 of the EPA guidance document)

0.126 = Conversion factor, lb/hr to g/s

The controlled emissions of acid gas is then estimated using Equation (10) from the EPA Screening Procedures:

$$ER_{Ac} = (ER_{Au}) \left[1 - \frac{CE_s}{100} \right]$$

Where:

ER_{Ac} = Controlled emission rate of acid gas, g/s

ER_{Au} = Uncontrolled emission rate of acid gas, g/s

CE_s = Scrubber control efficiency for the acid gas, %

Based on the EPA Screening Procedures, pages 24 and 25, a typical HCl control efficiency for a wet scrubber is 99%.

The above equations for each chlorine-containing compound can be simplified as follows to estimate average and maximum HCl controlled emission rates knowing: MW_A = 35.5 g/mole, FR = 120,000 lb/hr, C_{Ai} is in ppb, R_A = 1.028, and CE_s = 99%.

$$ER_{Ac} = (120,000)(CA_i) \left[\frac{35.5 \ N_{Ai}}{MW_{Ai}} \right] (1 \times 10^{-9})(1.028)(0.126) \left[1 - \frac{99}{100} \right]$$

$$ER_{Ac} = (5.517 \times 10^{-6})(C_{Ai}) \left[\frac{N_{Ai}}{MW_{Ai}} \right]$$

The ER_{Ac} for each compound is summed to determine the total controlled HCl emission rate for the Drake Chemical incinerator system.

In order to ensure that the risk assessment is based on conservatively high values, another approach was also taken to estimate an upper bound of HCl emissions considering all other acid gas forming compounds that potentially could be present in the feed stream. The upper bound HCl emission rate was assumed to be 4 lb/hr, the regulatory limit on HCl emissions from hazardous waste incinerators.

3.3.4 Particulate Matter

Particulate matter emissions can be estimated using Equation (11) from the EPA Screening Procedure:

$$ER_{PM} = (0.08 \text{ gr/dscf})(Q_G)(0.00108)$$

Where:

ER_{PM} = Emission rate of PM, g/s

Q_G = Gas flow rate, dry standard cubic feet per minute (dscfm) at 7% oxygen in the stack gas.

0.00108 = Conversion factor, gr/min to g/s

The assumed stack concentration of 0.08 gr/dscf at 7% oxygen is the maximum allowable limit of particulate matter per current RCRA standards for such an incinerator system.

EPA is currently developing revised RCRA regulations that will reduce the allowable particulate matter concentration to 0.015 gr/dscf at 7% oxygen. EPA policy requires newly permitted incinerators to meet this new limit (Gross, 1995). Therefore, particulate matter emissions were estimated for both concentrations.

It is conservatively assumed that particulate matter less than 10 micrometers aerodynamic diameter is equal to the total particulate matter calculated according to the procedure noted above.

3.3.5 Carbon Monoxide

Carbon monoxide (CO) emissions can be estimated from the concentration of 100 parts per million dry volume (ppmdv) at 7% oxygen suggested in EPA standards development documents (MRI, 1988). This value represents an indicator of good combustion that will limit emissions of products of incomplete combustion (PICs). This suggestion was also cited

in the Trial Burn Plan for the Drake Chemical Superfund Site's Mobile Hazardous Waste Incinerator (MRI, 1994).

$$ER_{co} = (100\text{ppmdv@7\%}O_2)Q_G(\text{dscf@7\%}O_2/\text{min}) \ \frac{cf/cf}{10^6\text{ppm}}$$

$$(lb-mole/385.3scf)(28.01lbCO/lb-mole)$$

$$(453.6g/lb)(\text{min}/60\text{sec})$$

$$= (100ppmdv)Q_G(5.49x10^{-7})$$

Where:

ER_{CO} = Emission rate of CO, g/s

Q_G = Gas flow rate, dry standard cubic feet per minute (dscfm) at 7% oxygen in the stack gas.

5.49×10^{-7} = Conversion factor

It should be noted that the Trial Burn plan (MRI, 1994) also cites a maximum instantaneous CO concentration of 500 ppmdv at 7% oxygen based on the same EPA guidance document.

3.3.6 Nitrogen Oxides

Formation of nitrogen oxide (NO_x) emissions is rather complex and thus not easily quantifiable. NO_x is generated in two ways:

- Fuels NO_x derived from the oxidation of nitrogen in the soils and organic and inorganic compounds in the waste feed as well as in the fuel; and

- Thermal NO_x is derived from the oxidation of nitrogen in the combustion air at relatively high temperature.

Therefore, NO_x formation is a function of many factors, including waste feed rate and nitrogen content, fuel feed rate and nitrogen content, excess air (available oxygen and nitrogen in the gas), flame temperature, burner design, and combustion chamber temperature and residence time. Because of this complexity no technique is available to estimate NO_x emissions accurately except by basing it on emission testing of similar combustion units. Based on experience with similar hazardous waste incinerators and waste feeds, the Trial Burn Plan (MRI, 1995) estimated the daily average NO_x emissions as 300 ppmdv at 7% oxygen; conservatively assuming all NO_x is nitrogen dioxide:

$$ER_{NO_x} = (300ppmdv@7\%O_2)Q_G(dscf@7\%O_2/min)\ \frac{cf/cf}{10^6ppm}$$

$$(lb-mole/385.3scf)(46.01lbNO_2/lb-mole)$$

$$(453.6g/lb)(min/60sec)$$

$$= (300ppmdv)Q_G(9.02x10^{-7})$$

Where:

ER_{co} = Emission rate of NO_x, g/s

Q_G = Gas flow rate, dry standard cubic feet per minute (dscfm) at 7% oxygen in the stack gas.

9.02x10-7 = Conversion factor

3.4   CALCULATIONS/TABLES OF EMISSION RESULTS

Tables 3-1 through 3-5 provide the raw data and calculated emissions rates for volatile organics, semi-volatile organics, metals, HCl and other criteria pollutants (PM/PM$_{10}$,CO, NO$_x$) respectively.

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TABLE 3-1
DRAKE CHEMICAL SITE INCINERATOR SYSTEM
ESTIMATED VOC EMISSION RATES

Compound	Feed Concentration C_o (µg/kg)		Removal Efficiency[b]	Estimated Emission Rate, ER_o (g/s)[c]	
	Maximum	Average		Maximum	Average
Benzene[a]	3.0	3.0	99.99%	4.54E-09	4.54E-09
2-Butanone[a]	1,400	190.6	99.99%	2.12E-06	2.88E-07
Carbon Tetrachloride[d]	17,000	670	99.99%	2.57E-05	1.01E-06
Chlorobenzene[a]	20,176	868.4	99.99%	3.05E-05	1.31E-06
Chloroform[a]	79.0	41.5	99.99%	1.19E-07	6.27E-08
1,2-Dichloroethane[a]	248.0	29.5	99.99%	3.75E-07	4.46E-08
1,1-Dichloroethene[d]	17,000	670	99.99%	2.57E-05	1.01E-06
1,1,2,2-Tetrachloroethane[d]	17,000	670	99.99%	2.57E-05	1.01E-06
Tetrachloroethylene[a]	38.0	23.8	99.99%	5.75E-08	3.60E-08
Toluene[a]	79.0	15.6	99.99%	1.19E-07	2.35E-08
1,1,1-Trichloroethane[a]	43.0	43.0	99.99%	6.50E-08	6.50E-08
1,1,2-Trichloroethane[d]	17,000	670	99.99%	2.57E-05	1.01E-06
Trichloroethylene[a]	2.0	2.0	99.99%	3.02E-09	3.02E-09
Vinyl Chloride[d]	17,000	670	99.99%	2.57E-05	1.01E-06

[a]From Table 4-5, Test Pit Analysis (Fixed Base) For Organic Compounds Drake Chemical Site-Phase III RI.

[b]Screening Procedures for Estimating The Air Impacts of Incineration at Superfund Sites, OAQPS, Air/Superfund National Technical Guidance Study Series, Report ASF-23, p. 16.

[c]ER_o, g/s = 60 ton/hr x hr/3600 sec x 2000 lb/ton x 453.6 g/lb x kg/1000g x g/1,000,000 µg x (1 0.9999) x C_o [µg/kg] = $(1.512 \times 10^{-9})(C_o, µg/kg)$.

[d]Feed concentrations from Test Pit Excavation Report, Drake Chemical Superfund Site, Lock Haven, Pennsylvania, Montgomery Watson, October 1994.

TABLE 3-2
DRAKE CHEMICAL SITE INCINERATOR SYSTEM
ESTIMATED SEMI-VOLATILE ORGANIC EMISSION RATES

Compound	Feed Concentration C_o (μg/kg)		Removal Efficiency[b]	Estimated Emission Rate, ER$_o$ (g/s)[c]	
	Maximum	Average		Maximum	Average
Benzo[a]anthracene[a]	42,000	6,933.33	99.99%	6.35E-05	1.05E-05
Benzo[b]fluoranthene[a]	41,000	14,140	99.99%	6.20E-05	2.14E-05
Benzo[k]fluoranthene[a]	29,000	11,565	99.99%	4.38E-05	1.75E-05
Benzo[a]pyrene[a]	34,000	13,605	99.99%	5.14E-05	2.06E-05
Chrysene[a]	52,000	19,292.5	99.99%	7.86E-05	2.92E-05
Dibenzo[a,h]anthracene[a]	11,000	11,000	99.99%	1.66E-05	1.66E-05
Indeno[1,2,3-cd]pyrene[a]	22,000	10,870	99.99%	3.33E-05	1.64E-05
Naphthalene[a]	7,000	1,295.5	99.99%	1.06E-05	1.96E-06
Pyrene[a]	100,000	37,742.5	99.99%	1.51E-04	5.71E-05
2,4-Dinitrotoluene[a]	120,000	7,653	99.99%	1.81E-04	1.16E-05
2,6-Dinitrotoluene[a]	120,000	7,653	99.99%	1.81E-04	1.16E-05
Nitrobenzene[a]	9,900	5,060	99.99%	1.50E-05	7.65E-06
1,2-Dichlorobenzene[a]	100,000	17,801	99.99%	1.51E-04	2.69E-05
1,4-Dichlorobenzene[a]	12,000	3,250.83	99.99%	1.81E-05	4.92E-06
Hexachlorobenzene[d]	120,000	7,620	99.99%	1.81E-04	1.15E-05
Pentachlorophenol[a]	130,000	44,166.67	99.99%	1.97E-04	6.68E-05
1,2,4-Trichlorobenzene[a]	21,000	4,268.5	99.99%	3.18E-05	6.45E-06
2,4,5-Trichlorophenol[d]	310,000	19,521	99.99%	4.69E-04	2.95E-05
2,4,6-Trichlorophenol[a]	13,000	13,000	99.99%	1.97E-05	1.97E-05
β-Napthylamine[a]	220,000	88,624.44	99.99%	3.33E-04	1.34E-04
4-Nitroaniline[a]	48,000	48,000	99.99%	7.26E-05	7.26E-05
2-Nitroaniline[d]	310,000	18,745	99.99%	4.69E-04	2.83E-05
2,3,7,8-TCDD-TEQ (Dioxin)	-	-	-	30E-09[e]	30E-09[e]

[a]From Table 4-5. Test Pit Analysis (Fixed Base) For Organic Compounds Drake Chemical Site-- Phase III RI.

[b]Screening Procedures for Estimating The Air Impacts of Incineration at Superfund Sites. OAQPS. Air/Superfund National Technical Guidance Study Series. Report ASF-23. p. 16.

[c]ER$_o$, g/s = 60 ton/hr x hr/3600 sec x 2000 lb/ton x 453.6 g/lb x kg/1000g x g/1,000,000 μg x (1-0.9999) x C_o [μg/kg] = (1.512 x 10^{-9})(C_o, μg/kg)

[d]Feed concentrations from Test Pit Excavation Report. Drake Chemical Superfund Site. Lock Haven. Pennsylvania. Montgomery Watson. October 1994.

[e]Based on direction from EPA.

TABLE 3-3
DRAKE CHEMICAL SITE INCINERATOR SYSTEM
ESTIMATED METALS EMISSION RATES

Metal	Feed Concentration C_m (mg/kg)[a]		Partition Factor PF	Removal Efficiency[c] CE_m	Estimated Emission Rate, ER_{mc} (g/s)[e]	
	Maximum	Average			Maximum	Average
Arsenic	21	7.34	100 [b]	90%	3.18E-02	1.11E-02
Barium	382	111.65	50 [b]	95%	1.44E-01	4.22E-02
Beryllium	2.3	1.1	5 [b]	95%	8.69E-05	4.16E-05
Cadmium	283	33.94	100 [b]	90%	4.28E-01	5.13E-02
Chromium (III, VI)	269	49.54	5 [b]	95%	1.02E-02	1.87E-03
Lead	1170	63.12	100 [b]	90%	1.77E+00	9.54E-02
Mercury	18	2.54	100 [b]	50%[d]	1.36E-01	1.92E-02
Nickel	41	17.48	100 [f]	92.5%[g]	4.65E-02	1.98E-02
Selenium	1.6	1.6	100 [f]	70%[h]	7.26E-03	7.26E-03
Silver	3.8	3.8	100 [b]	95%	2.87E-03	2.87E-03

[a]From Table 4-5, Test Pit Analysis (Fixed Base) For Organic Compounds Drake Chemical Site—Phase III RI.

[b]Screening Procedures for Estimating The Air Impacts of Incineration at Superfund Sites, U.S. EPA, OAQPS, Air/Superfund National Technical Guidance Study Series, Report ASF-23, February 1992, p. 20; used 1600°F since the Drake Chemical rotary incinerator will be operating at 1000°F in the kiln and 1800°F in the secondary combustion chamber; Cl less than 1% of total feed by weight (refer

[c]Ibid. p. 22 for control by fabric filter and wet scrubber.

[d]Ibid, p.22 - footnote (a) of Table 6 states that for the indicated mercury removal for fabric filter/wet scrubber (FF/WS) to be valid, it is assumed that flue gases have been precooled (usually in a quench). The Drake Chemical incinerator system contains a water quench prior to the FF/WS, therefore, the mercury removal efficiency can be assumed to be valid.

[e]ER_{mc}, g/s = 60 ton/hr x hr/3600 sec x 2000 lb/ton x 453.6 g/lb x kg/1000g x g/1000 mg x (PF/100) x (1-CE_m/100) x C_m [mg/kg] = (1.512 x 10^5)(C_m, mg/kg)(PF)(100-CE_m).

[f]No data is given for a nickel or selenium partition factor in the EPA Screening Procedures. Therefore, to be conservative, it was assumed that all nickel and selenium present in the soil will be partitioned to the air pollution control devices. Data on the volatilized fraction in the Mitre Corp. report, Hazardous Waste Stream Trace Metal Concentrations and Emissions, suggests that the partitioning of these two compounds will be similar to those with a 100% partition factor.

[g]Based on relative electrostatic precipitator (ESP)/scrubber system removal from report by Mitre Corp., Hazardous Waste Stream Trace Metal Concentrations and Emissions, U.S. EPA Office of Solid Waste, November 1983 - assumed between arsenic (90%) and chromium (95%).

[h]Based on relative electrostatic precipitator (ESP)/scrubber system removal from Hazardous Waste Stream Trace Metal Concentrations and Emissions, Mitre Corp., U.S. EPA Office of Solid Waste, November 1983, assumed between mercury (50%) and antimony (90%) taking removal efficiencies from Screening Procedures for Estimating the Air Impacts of Incinerator of Superfund Sites, U.S. EPA OAQPS, Air/Superfund National Technical Guidance Study Series, Report ASF-23, February 1992.

TABLE 3-4
DRAKE CHEMICAL SITE INCINERATOR SYSTEM
ESTIMATED HCl ACID GAS EMISSION RATES

Compound	Feed Concentration C_{xi} (µg/kg)		Molecular Weight MW_{xi} (g/mole)	Moles Cl Atoms per Mole N_{xi} (mol Cl/mol)	Percent Chlorine by Weight in Soil		Estimated Uncontrolled HCl Emission Rate, ER_{xu} (g/s)		Removal Efficiency CE_x	Estimated Controlled HCl Emission Rate, ER_{xc} (g/s)	
	Maximum	Average			Maximum	Average	Maximum	Average		Maximum	Average
Benzene[a]	3	3	78.11	0	0	0	0.00E+00	0.00E+00	99%	0.00E+00	0.00E+00
2-Butanone[a]	1,400	190.6	72.1	0	0	0	0.00E+00	0.00E+00	99%	0.00E+00	0.00E+00
Carbon Tetrachloride[a]	17,000	670	153.82	4	0.0015607%	0.0000621%	2.44E-01	9.60E-03	99%	2.44E-03	9.60E-05
Chlorobenzene[a]	20,176	868.38	112.56	1	0.0000635%	0.0000027%	9.88E-04	4.25E-03	99%	9.88E-04	4.25E-05
Chloroform[a]	79	41.5	119.39	3	0.0000007%	0.0000004%	1.09E-05	5.75E-04	99%	1.09E-05	5.75E-06
1,2-Dichloroethane[a]	248	29.5	98.96	2	0.0000188%	0.0000020%	2.76E-03	3.29E-04	99%	2.76E-05	3.29E-06
1,1-Dichloroethene[a]	17,000	670	96.95	2	0.0012433%	0.0000049%	1.93E-01	7.62E-03	99%	1.93E-03	7.62E-05
1,1,2,2-Tetrachloroethane[a]	17,000	670	167.85	4	0.0014365%	0.0000057%	2.23E-01	8.80E-03	99%	2.23E-03	8.80E-05
Tetrachloroethylene[a]	38	23.8	165.83	4	0.0000035%	0.0000022%	5.05E-04	3.10E-04	99%	5.05E-06	3.10E-06
Toluene[a]	79	15.36	92.14	0	0	0	0.00E+00	0.00E+00	99%	0.00E+00	0.00E+00
1,1,1-Trichloroethane[a]	43	43	133.40	3	0.0000035%	0.0000035%	5.33E-04	5.33E-04	99%	5.33E-06	5.33E-06
1,1,2-Trichloroethane[a]	17,000	670	133.40	3	0.0015944%	0.0000063%	2.48E-01	9.77E-03	99%	2.48E-03	9.77E-05
Trichloroethylene[a]	2	2	131.39	3	0.0000002%	0.0000002%	2.52E-05	2.52E-05	99%	2.52E-07	2.52E-07
Vinyl Chloride[a]	17,000	670	62.50	1	0.0009043%	0.0000389%	1.50E-03	5.91E-03	99%	1.50E-03	5.91E-05
Benzo[a]anthracene[a]	42,000	6,933.33	278.33	0	0	0	0.00E+00	0.00E+00	99%	0.00E+00	0.00E+00
Benzo[b]fluoranthene[a]	41,000	14,140	252.32	0	0	0	0.00E+00	0.00E+00	99%	0.00E+00	0.00E+00
Benzo[k]fluoranthene[a]	29,000	11,365	252.32	0	0	0	0.00E+00	0.00E+00	99%	0.00E+00	0.00E+00
Benzo[a]pyrene[a]	34,000	13,605	252.30	0	0	0	0.00E+00	0.00E+00	99%	0.00E+00	0.00E+00
Chrysene[a]	52,000	19,292.5	228.29	0	0	0	0.00E+00	0.00E+00	99%	0.00E+00	0.00E+00
Dibenzo[a,h]anthracene[a]	11,000	11,000	278.33	0	0	0	0.00E+00	0.00E+00	99%	0.00E+00	0.00E+00
Indeno[1,2,3-cd]pyrene[a]	22,000	10,870	276.00	0	0	0	0.00E+00	0.00E+00	99%	0.00E+00	0.00E+00
Naphthalene[a]	7,000	1,295.5	128.17	0	0	0	0.00E+00	0.00E+00	99%	0.00E+00	0.00E+00
Pyrene[a]	100,000	37,742.5	202.26	0	0	0	0.00E+00	0.00E+00	99%	0.00E+00	0.00E+00
1,3-Dinitrobenzene[a]	NA	NA	168.11	0	0	0	0.00E+00	0.00E+00	99%	0.00E+00	0.00E+00
2,4-Dinitrotoluene[a]	120,000	7,653	182.14	0	0	0	0.00E+00	0.00E+00	99%	0.00E+00	0.00E+00
2,6-Dinitrotoluene[a]	120,000	7,653	182.14	0	0	0	0.00E+00	0.00E+00	99%	0.00E+00	0.00E+00
Nitrobenzene[a]	130,000	44,166.67	266.34	6	0.0086532%	0.0029399%	1.35E+00	4.57E-01	99%	1.35E-02	4.57E-03
Pentachlorobenzene[a]	9,900	5,060	250.34	5	0.0012311%	0.0002508%	1.91E-01	3.89E-02	99%	1.91E-02	3.89E-04
1,2-Dichlorobenzene[a]	NA	NA	147.00	2	0.0048233%	0.0008559%	7.50E-01	1.33E-01	99%	7.50E-03	1.33E-03
1,4-Dichlorobenzene[a]	100,000	17,401	147.00	2	0.0003799%	0.0001157%	9.00E-02	2.44E-02	99%	9.00E-04	2.44E-04
Hexachlorobenzene[a]	12,000	3,250.83	284.78	6	0.0089631%	0.0006069%	1.39E+00	8.85E-02	99%	1.39E-02	8.85E-04
Pentachlorophenol[a]	120,000	7,620	266.34	5	0.0069471%	0.0002039%	1.35E+00	4.57E+00	99%	1.35E-02	4.57E-03
1,2,4-Trichlorobenzene[a]	21,000	4,268.5	181.45	3	0.0010691%	0.0002039%	1.91E-01	3.89E-02	99%	1.91E-03	3.89E-04
2,4,5-Trichlorophenol[a]	310,000	19,321	197.45	3	0.0106971%	0.0010103%	2.00E-01	1.63E-02	99%	2.00E-02	1.63E-03
2,4,6-Trichlorophenol[a]	13,000	13,000	197.45	3	0.0007005%	0.0007005%	1.09E-01	1.09E-01	99%	1.09E-03	1.09E-03
β-Naphthylamine[a]	220,000	88,624.44	143.19	0	0	0	0.00E+00	0.00E+00	99%	0.00E+00	0.00E+00
4-Nitroaniline[a]	48,000	48,000	138.13	0	0	0	0.00E+00	0.00E+00	99%	0.00E+00	0.00E+00
2-Nitroaniline[a]	310,000	18,745	138.13	0	0	0	0.00E+00	0.00E+00	99%	0.00E+00	0.00E+00
Total					0.0493%	0.0047%	7.636	1.062		0.076	0.011

[a] Feed concentrations from Table 4-5, Test Pit Analysis (Fixed Base) For Organic Compounds Drake Chemical Site--Phase III R1

[b] Feed concentrations from Test Pit Excavation Report, Drake Chemical Superfund Site, Lock Haven, Pennsylvania, Montgomery Watson, October 1994

[c] ER_{xu}, g/s = 60 ton/hr x hr/3600 sec x 2000 lb/ton x 453.6 g/lb x kg/1000 g x C_{xi} [µg/kg] x g/1,000,000 µg x MW_{xi} [mol/g] x N_{xi} x 36.45 g HCl/mol Cl

[d] Hydrogen chloride emissions are controlled by a packed bed scrubber - from Screening Procedures for Estimating The Air Impacts of Incineration at Superfund Sites, OAQPS, Au/Superfund National Technical Guidance Study Series, Report ASF-23, p. 24, wet scrubber control efficiency for HCl is typically 99%

[e] ER_{xc}, g/s = 60 ton/hr x hr/3600 sec x 2000 lb/ton x 453.6 g/lb x kg/1000 g x C_{xi} [µg/kg] x g/1,000,000 µg x MW_{xi} [mol/g] x N_{xi} x 36.45 g HCl/mol Cl x (1-CE_x) = (3.517 x 10^{-6} µg/kg $\frac{N_{xi}}{MW_{xi}}$ mol/g)

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TABLE 3-5
DRAKE CHEMICAL SITE INCINERATOR SYSTEM
ESTIMATED PM, CO, AND NO_x EMISSION RATES

I. PARTICULATE MATTER (PM)
From <u>Screening Procedures for Estimating the Air Impacts of Incineration at Superfund Site</u>, OAQPS, Air/Superfund National Technical Guidance Study Series. Report ASF-23, p. 25

$$ER_{PM} = (0.08 \text{ gr/dscf})(Q_G)(0.00108)$$

ER_{PM} = Emission rate of particulate matter (PM), grams/second (g/s)

Q_G = Gas flow rate, dry standard cubic feet per minute (dscfm) at 7% oxygen in the stack gas

0.00108 = Conversion factor, grains/minute (gr/min) to g/s

From Section 2 of this report, Q_G = 50,000 dscfm at 7% oxygen.

$$ER_{PM} = 4.32 \text{ g/s}$$

According to current EPA RCRA policy PM emissions must be limited to 0.015 gr/dscf at 7% oxygen.

$$ER_{PM} = 0.81 \text{ g/s}$$

It is conservatively assumed that $ER_{PM10} = ER_{PM}$.

II. CARBON MONOXIDE (CO)
From <u>Guidance on Carbon Monoxide Controls for Hazardous Waste Incinerators</u>, MRI Draft Final Report for EPA Office of Solid Waste, p. 1-1, maximum hourly average carbon monoxide (CO) concentration is assumed to be 100 ppmdv at 7% oxygen.

$$ER_{CO} = (100 \text{ ppmdv})(Q_G)(5.49 \times 10^{-7})$$

As noted above, Q_G = 50,000 dscfm at 7% oxygen.

$$ER_{CO} = 2.75 \text{ g/s}$$

III. NITROGEN OXIDES (NO_x)
Based on the <u>Trial Burn Plan for the Drake Chemical Superfund Site's Mobile Hazardous Waste Incinerator</u>, the daily average NO_x concentration is assumed to be 300 ppmdv at 7% oxygen.

$$ER_{NOx} = (300 \text{ ppmdv})(Q_G)(9.02 \times 10^{-7})$$

As noted above, Q_G = 50,000 dscfm at 7% oxygen.

$$ER_{NOx} = 13.5 \text{ g/s}$$

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SECTION 4

AIR QUALITY IMPACT ANALYSIS

4.1 INTRODUCTION

This section presents the air quality impacts associated with the emissions during the trial burn of the proposed incinerator at the Drake Chemical Site. Specifically, the following items were addressed:

- The modeling approach and procedures used to identify air quality impacts associated with the proposed trial burn.

- The predicted pollutant concentrations due to the emissions from the incinerator during the trial burn.

The ambient air quality modeling analysis performed by WESTON was based on information provided by U.S. EPA Region III, and originally developed by Montgomery Watson and Midwest Research Institute (MRI, 1993). The information provided included a model-ready input file including all source characteristics, a receptor grid including terrain elevations, and building downwash information. Also provided was a meteorological data file from the on-site meteorological station (for a one-year period beginning in November 1992).

The following subsections describe WESTON's modeling analysis using the information generated by MRI.

4.2 LAND USE CLASSIFICATION AND TOPOGRAPHY

The nature of the land use in the area surrounding the Drake Chemical Site in Lock Haven, PA was determined using the Auer technique as outlined in Auer (1978) and EPA (1994c). The designation of the area as urban or rural determines which dispersion coefficients are used in the modeling analysis. Factors which affect atmospheric dispersion and the

designation of land use as urban or rural include: the types of industry and commerce, building types, extent of vegetated surface area, and water surface area.

The Auer (1978) technique establishes four primary land use types: industrial; commercial; residential; and agricultural. Industrial, commercial, and compact residential areas are classified as urban, while agricultural and common residential areas are considered rural. For modeling purposes, an area is defined as urban when more than 50 percent of the surface area within 3 kilometers of the source falls under an urban land use type. Otherwise, the area is determined to be rural.

The location of the Drake Chemical Site was plotted on a 7.5 minute United States Geologic Survey (USGS) map (1:24,000 scale). A 3-kilometer radius circle was drawn around the facility, and land use was then assessed within the circle. Based on inspection, greater than 50% of the surrounding area was rural. Therefore, the region surrounding the site was classified as rural for air quality modeling purposes, and rural diffusion coefficients were used in the modeling analysis.

Based on the topography of the area near the Drake Chemical site, the terrain elevation was below the height of the stack in some areas, and in other areas the terrain elevation exceeded both the stack height and final plume height. As a result, a model which estimates concentrations for both simple and complex terrain receptors was used in the modeling analysis. Simple terrain receptors are defined by EPA (1994c) as those having elevations which are below stack top, and complex terrain receptors are defined as those lying above the final plume height. Both simple and complex terrain model algorithms were used in areas which EPA designates as intermediate terrain, where elevations fall between the stack top and final plume height. Each of these model algorithms for estimating ambient concentration are described below.

4.3   MODEL FOR INHALABLE CONCENTRATION

The Industrial Source Complex (ISC3) (Version 95200) short-term dispersion model was used to estimate ambient concentrations in simple and complex rural terrain locations. The ISC3 model is a U.S. EPA-approved model. The model provides options to predict concentrations from a wide range of sources. The basis of the model is the straight-line, steady-state Gaussian plume equation, which is used with some modifications to model simple point source emissions from stacks; emissions from stacks that experience the effects of aerodynamic downwash from nearby buildings; isolated vents; multiple vents; storage piles; and conveyor belts. The ISC3 short-term model accepts hourly meteorological data to define the conditions for plume rise, transport, and dispersion. The model estimates the concentration or deposition value for each source and receptor combination for each hour of entered meteorological conditions and calculates user-selected short-term averages. Concentration estimates are made for terrain elevations up to stack top using the ISCST algorithms. For elevations above stack top, the ISC3 model uses the COMPLEX I/Valley calculation algorithms. For terrain elevations between stack top and plume height (intermediate terrain), the ISC3 model will select the higher of the simple and complex terrain calculations on an hour-by-hour, source-by-source, and receptor-by-receptor basis.

The selection of an appropriate air dispersion model depends on the ability of the model to simulate the dispersive conditions that potentially affect the source to be evaluated. For the Drake Chemical site, areas of elevated terrain are present which required that the air dispersion modeling approach incorporate models that U.S. EPA has recommended for elevated terrain (i.e., complex terrain) areas. Possible air dispersion models include ISCST3 CTDMPLUS/CTSCREEN, AERMOD, and CALPUFF. Of the four models, ISCST3 and CALPUFF were applied at the Drake Chemical site.

The dispersion algorithms that the ISCST3 and CALPUFF models use to simulate the dispersion of pollutants differ in many areas. The most basic difference involves the treatment of plume transport and dispersion. Other differences are elevated terrain (complex terrain) and the ability of the CALPUFF model to incorporate vertical wind shear.

The CALPUFF model has been utilized by U.S. EPA to determine the ambient concentrations due to the proposed incinerator during stagnation periods or near calm wind conditions. The preliminary modeling using the CALPUFF model indicates the ambient concentrations during stagnation periods are lower than the concentrations predicted by the ISC3 model. Therefore, by utilizing the ISCST3 model, conservative estimates of the ambient concentrations were assumed and used in the risk assessment.

4.4 RECEPTOR GRID

A polar coordinate system with 22 rings and 36 radials and centered upon the location of the incinerator stack of the Drake Chemical Site was used as a basis for receptor deployment for the ISC3 model. The 36 radials were evenly spaced at 10-degree intervals beginning with North, and the grid origin was located at the incinerator stack. The 22 radials were spaced at 50-meter intervals out to 100 m; at 100-meter intervals out to 3,000 m; and at 200-meter intervals out to 3,200 meters. This grid, including elevation data, was developed by MRI. WESTON used desktop mapping software to translate the receptors into geographical coordinates, and contour the elevation data. The contoured model grid elevation patterns compared well with the USGS topographical maps of the area. Based on the translation, the approximate Universal Transverse Mercator (UTM) coordinates of the incinerator stack location (the grid origin) were determined to be 294.87 km Easting, 4,556.05 km Northing, Zone 18.

In addition to the main polar receptor grid, MRI included additional receptors to represent the site boundary and sensitive receptors (schools and hospitals). The site boundary was represented by 36 receptors added along the 36 main polar grid radials. A total of 12 sensitive receptors were identified by MRI. Based on a survey of the USGS topographical maps, WESTON identified these sensitive receptors as the following:

- Dickey School
- Immaculate Conception School
- Lock Haven Hospital

- Lock Haven Junior/Senior High School
- Lock Haven University of Pennsylvania
- McGhee School
- Penn School
- Robb School
- Saint Agnes School
- Unidentified School (in Castanea)
- Unidentified School (in Flemington)
- Woodward School (near Dunnstown)

Except as noted, all of these sites are located in Lock Haven. Because the risk assessment evaluates maximum predicted concentrations and maximum exposure scenarios, separate risk evaluations for these receptors were not performed.

4.5   METEOROLOGICAL DATA

The meteorological database for the ISC3 model consisted of surface data collected at the Drake Chemical Site. A 3-month period (December 1992 through February 1993) of meteorological data was used in the modeling to represent the conditions most likely to be experienced during the trial burn. A windrose for the three month period used in the modeling is presented in Figure 4-1. A consistent average daily winter mixing height of 800 m based on data from Holzworth (1972) was used in the modeling.

4.6   MODEL OPTIONS

The ISC3 model has various options to simulate different dispersion conditions for emissions from a stack. The U.S. EPA has recommended various options to be used in dispersion modeling for regulatory purposes. These recommended regulatory default options, shown in Table 4-1, were used in the air quality impact analysis for the trial burn.

FIGURE 4-1

DRAKE CHEMICALS SITE METEOROLOGICAL STATION
LOCK HAVEN, PENNSYLVANIA
WINTER SEASON (01 DEC 1992 - 28 FEB 1993)
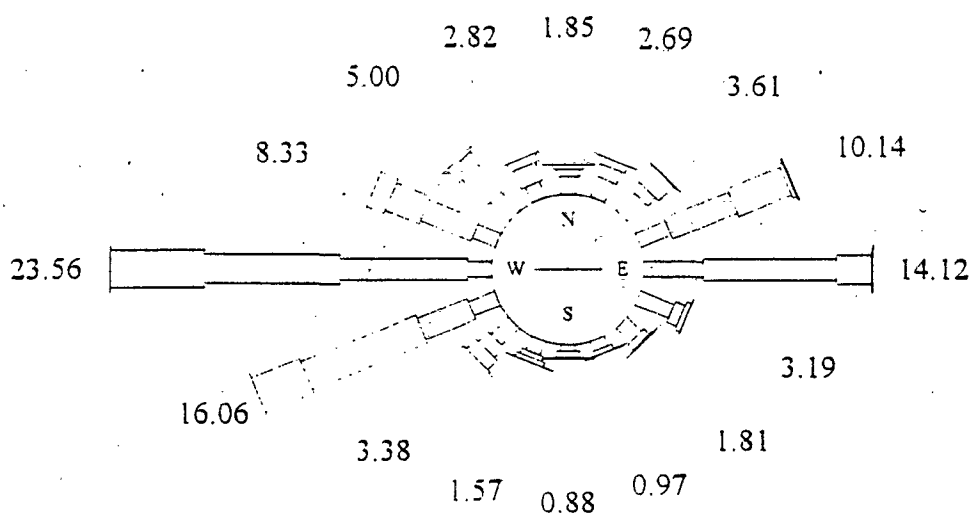
SCALE (M/SEC)

WIND SPEED (M/SEC)	PERCENT OCCURRENCE					
	0-1.3	1.3-3.0	3.0-5.1	5.1-8.2	8.2-10.8	10.8
N	1.06	0.51	0.28	0.00	0.00	0.00
NNE	1.34	0.83	0.51	0.00	0.00	0.00
NE	1.57	0.69	1.30	0.05	0.00	0.00
ENE	1.99	4.31	3.52	0.23	0.09	0.00
E	3.66	8.19	2.18	0.05	0.05	0.00
ESE	2.22	0.65	0.32	0.00	0.00	0.00
SE	1.25	0.46	0.09	0.00	0.00	0.00
SSE	0.65	0.32	0.00	0.00	0.00	0.00

WIND SPEED (M/SEC)	PERCENT OCCURRENCE					
	0-1.3	1.3-3.0	3.0-5.1	5.1-8.2	8.2-10.8	10.8
S	0.42	0.42	0.05	0.00	0.00	0.00
SSW	0.93	0.32	0.32	0.00	0.00	0.00
SW	1.53	0.88	0.83	0.14	0.00	0.00
WSW	1.76	3.33	7.73	3.24	0.00	0.00
W	1.48	7.92	8.38	5.74	0.05	0.00
WNW	1.53	3.38	2.27	1.16	0.00	0.00
NW	1.67	2.22	0.93	0.19	0.00	0.00
NNW	1.06	1.20	0.56	0.00	0.00	0.00

TABLE 4-1

REGULATORY DEFAULT MODEL OPTIONS

ISCST MODEL (SIMPLE TERRAIN)

- Stack-tip downwash

- Final plume rise.

- Buoyancy induced dispersion (BID).

- Default Vertical potential temperature gradients.

- Automatic treatment of calms.

- Default wind profile exponents.

- Infinite pollutant half-life.

- No missing data processing.

- Calculate "upper bound" values for supersquat buildings.

COMPLEX I MODEL (COMPLEX TERRAIN)

- Use Terrain Adjustments.

- Use buoyancy induced dispersion.

- Plume heights are not allowed closer than ZMIN to receptors.

- ZMIN is set to 10 meters.

- Automatic treatment of calms.

- Terrain adjustments are: 0.5, 0.5, 0.5, 0.5, 0.0, 0.0 for stability classes A through F, respectively.

- Flag pole receptors can not be used.

4.7 EMISSIONS CHARACTERISTICS

The physical emission parameters for the proposed incinerator at the Drake Chemical Site are presented in Table 4-2. These physical emission parameters were used in the air quality modeling analysis and represent the expected operating conditions of the incinerator during the trial burn. The pollutant emission rates shown in Table 4-3 represent the maximum and average emission rates based on expected soil concentrations of the pollutants and the destruction removal efficiency of the incinerator, as described in Section 3. These potential emission estimates were used in the modeling analysis to determine the potential impact of the incinerator. Table 4-3 summarizes the data presented in Tables 3-1 through 3-5.

4.8 GOOD ENGINEERING PRACTICE ANALYSIS

Section 123 of the federal Clean Air Act (CAA) defines Good Engineering Practice (GEP), with respect to stack heights, as "the height necessary to ensure that emissions from the stack do not result in excessive concentrations of any pollutant in the immediate vicinity of the source as a result of atmospheric downwash, eddies, or wakes which may be created by the source itself, nearby structures, or nearby terrain obstacles."

According to 40 CFR 51.1(ii), GEP stack height is calculated by the relationship:

$$Hg = H + 1.5 \, L$$

Where:

Hg = GEP stack height

H = height of nearby structure(s) measured from the ground level elevation at the base of the stack.

L = lesser of height or projected width of nearby structures.

A stack whose height is greater than or equal to this formula GEP height will not be affected by building wake-effect induced downwash.

TABLE 4-2

PHYSICAL STACK CHARACTERISTICS
DRAKE CHEMICALS SITE
LOCK HAVEN, PENNSYLVANIA

STACK CHARACTERISTIC	UNITS	INCINERATOR
Stack Location	UTM Coordinates (m)	294,870 E 4,556,050 N
Stack Elevation	m	169.2
Stack Height	m	45.7
Stack Diameter	m	1.8
Gas Temperature	°K	359.0
Gas Exit Velocity	m/sec	16.4

TABLE 4-3
POLLUTANT EMISSION RATES USED IN AMBIENT AIR QUALITY MODELING
DRAKE CHEMICAL SITE

Category/ Compound	Estimated Emission Rate (g/s)	
	Maximum	Average
VOC		
Benzene	4.54E-09	4.54E-09
2-Butanone	2.12E-06	2.88E-07
Carbon Tetrachloride	2.57E-05	1.01E-06
Chlorobenzene	3.05E-05	1.31E-06
Chloroform	1.19E-07	6.27E-08
1,2-Dichloroethane	3.75E-07	4.46E-08
1,1-Dichloroethene	2.57E-05	1.01E-06
1,1,2,2-Tetrachloroethane	2.57E-05	1.01E-06
Tetrachloroethylene	5.75E-08	3.60E-08
Toluene	1.19E-07	2.35E-08
1,1,1-Trichloroethane	6.50E-08	6.50E-08
1,1,2-Trichloroethane	2.57E-05	1.01E-06
Trichloroethylene	3.02E-09	3.02E-09
Vinyl Chloride	2.57E-05	1.01E-06
TRACE METALS		
Arsenic	3.18E-02	1.11E-02
Barium	1.44E-01	4.22E-02
Beryllium	8.69E-05	4.16E-05
Cadmium	4.28E-01	5.13E-02
Chromium (III, VI)	1.02E-02	1.87E-03
Lead	1.77E+00	9.54E-02
Mercury	1.36E-01	1.92E-02
Nickel	4.65E-02	1.98E-02
Selenium	7.26E-03	7.26E-03
Silver	2.87E-03	2.87E-03
OTHER COMPOUNDS		
Carbon Monoxide	2.75E+00	2.75E+00
Hydrogen Chloride	7.44E-02	1.05E-02
Inhalable Particulates (PM-10)	8.10E-01	8.10E-01
Nitrogen Oxides	1.35E+01	1.35E+01

Category/ Compound	Estimated Emission Rate (g/s)	
	Maximum	Average
SEMI-VOLATILE ORGANICS		
Benzo[a]anthracene	6.35E-05	1.05E-05
Benzo[b]fluoranthene	6.20E-05	2.14E-05
Benzo[k]fluoranthene	4.38E-05	1.75E-05
Benzo[a]pyrene	5.14E-05	2.06E-05
Chrysene	7.86E-05	2.92E-05
Dibenzo[a,h]anthracene	1.66E-05	1.66E-05
Indeno[1,2,3-cd]pyrene	3.33E-05	1.64E-05
Naphthalene	1.06E-05	1.96E-06
Pyrene	1.51E-04	5.71E-05
1,3-Dinitrobenzene	NA	NA
2,4-Dinitrotoluene	1.81E-04	1.16E-05
2,6-Dinitrotoluene	1.81E-04	1.16E-05
Nitrobenzene	1.50E-05	7.65E-06
Pentachlorobenzene	NA	NA
1,2-Dichlorobenzene	1.51E-04	2.69E-05
1,4-Dichlorobenzene	1.81E-05	4.92E-06
Hexachlorobenzene	1.81E-04	1.15E-05
Pentachlorophenol	1.97E-04	6.68E-05
1,2,4-Trichlorobenzene	3.18E-05	6.45E-06
2,4,5-Trichlorophenol	4.69E-04	2.95E-05
2,4,6-Trichlorophenol	1.97E-05	1.97E-05
b-Napthylamine	3.33E-04	1.34E-04
4-Nitroaniline	7.26E-05	7.26E-05
2-Nitroaniline	4.69E-04	2.83E-05
2,3,7,8-TCDD TEQ (Dioxin)	3.00E-08	3.00E-08

Based on the downwash-effects building dimensions included in the MRI model input file, the dominant downwash structure for the incinerator stack is 55 feet tall, with a maximum projected width of 246 feet. The formula GEP height for this structure is 137.5 feet. The incinerator stack height of 150 feet (45.7 meters) is greater than the formula GEP height, and therefore downwash effects were not included in the model calculations.

4.9 PREDICTED AIR QUALITY IMPACTS

The maximum predicted ground-level air pollutant concentrations of metals and other pollutants, as estimated by the ISC3 model, are presented in Tables 4-4 and 4-5. The maximum normalized Chi/Q values are presented at the bottom of Table 4-4. The Chi/Q value, when multiplied by the emission rate for a specific pollutant, results in the maximum off-site ambient concentration for that pollutant. Chi/Q values have been developed with short-term (1-hour) and long-term (30-day) averages to represent different exposure periods in the risk calculations.

Table 4-4 presents the maximum estimated one-hour and thirty-day concentrations for volatile organic compounds (VOCs), semi-volatile organic compounds (SVOCs), trace metals and hydrogen chloride (HCl). These concentrations were used in the health risk assessment.

Table 4-5 presents the maximum concentrations for those pollutants for which a National Ambient Air Quality Standard (NAAQS) has been established. The table contains the maximum estimated quantity concentrations for lead, the maximum 24-hour concentration for inhalable particulate (PM_{10}), the maximum 1 and 8-hour concentrations for carbon monoxide (CO) and the maximum quarterly average for nitrogen dioxide (NO_x) since only three months of meterological data were used in the modeling.

As presented in Table 4-5, predicted concentrations for PM_{10}, Lead, CO, and NO_x are well below their associated NAAQS.

TABLE 4-4
MODELED MAXIMUM AMBIENT AIR IMPACTS
FOR SCREENING HEALTH RISK ASSESSMENT
DRAKE CHEMICAL SITE

Category/ Compound	Estimated Emission Rate (g/s) Maximum	Average	Maximum Modeled Impact (ug/m³) 1-Hour	30-Day
VOC				
Benzene	4.54E-09	4.54E-09	1.88E-07	2.02E-09
2-Butanone	2.12E-06	2.88E-07	8.77E-05	1.28E-07
Carbon Tetrachloride	2.57E-05	1.01E-06	1.06E-03	4.49E-07
Chlorobenzene	3.05E-05	1.31E-06	1.26E-03	5.82E-07
Chloroform	1.19E-07	6.27E-08	4.93E-06	2.79E-08
1,2-Dichloroethane	3.75E-07	4.46E-08	1.55E-05	1.98E-08
1,1-Dichloroethene	2.57E-05	1.01E-06	1.06E-03	4.49E-07
1,1,2,2-Tetrachloroethane	2.57E-05	1.01E-06	1.06E-03	4.49E-07
Tetrachloroethylene	5.75E-08	3.60E-08	2.38E-06	1.60E-08
Toluene	1.19E-07	2.35E-08	4.93E-06	1.04E-08
1,1,1-Trichloroethane	6.50E-08	6.50E-08	2.69E-06	2.89E-08
1,1,2-Trichloroethane	2.57E-05	1.01E-06	1.06E-03	4.49E-07
Trichloroethylene	3.02E-09	3.02E-09	1.25E-07	1.34E-09
Vinyl Chloride	2.57E-05	1.01E-06	1.06E-03	4.49E-07
TRACE METALS				
Arsenic	3.18E-02	1.11E-02	1.32E+00	4.93E-03
Barium	1.44E-01	4.22E-02	5.96E+00	1.88E-02
Beryllium	8.69E-05	4.16E-05	3.60E-03	1.85E-05
Cadmium	4.28E-01	5.13E-02	1.77E+01	2.28E-02
Chromium (III, VI)	1.02E-02	1.87E-03	4.22E-01	8.31E-04
Lead	1.77E+00	9.54E-02	7.33E+01	4.24E-02
Mercury	1.36E-01	1.92E-02	5.63E+00	8.58E-03
Nickel	4.65E-02	1.98E-02	1.92E+00	8.80E-03
Selenium	7.26E-03	7.26E-03	3.00E-01	3.23E-03
Silver	2.87E-03	2.87E-03	1.19E-01	1.28E-03
OTHER COMPOUNDS				
Hydrogen Chloride	7.44E-02	1.05E-02	3.08E+00	4.67E-03

	1-Hour	30-Day
MAXIMUM NORMALIZED CHI/Q, [(ug/m³)/(g/s)]	41.39	0.444

Category/ Compound	Estimated Emission Rate (g/s) Maximum	Average	Maximum Modeled Impact (ug/m³) 1-Hour	30-Day
SEMI-VOLATILE ORGANICS				
Benzo[a]anthracene	6.35E-05	1.05E-05	2.63E-03	4.67E-06
Benzo[b]fluoranthene	6.20E-05	2.14E-05	2.57E-03	9.51E-06
Benzo[k]fluoranthene	4.38E-05	1.75E-05	1.81E-03	7.78E-06
Benzo[a]pyrene	5.14E-05	2.06E-05	2.13E-03	9.15E-06
Chrysene	7.86E-05	2.92E-05	3.25E-03	1.30E-05
Dibenzo[a,h]anthracene	1.66E-05	1.66E-05	6.87E-04	7.38E-06
Indeno[1,2,3-cd]pyrene	3.33E-05	1.64E-05	1.38E-03	7.29E-06
Naphthalene	1.06E-05	1.96E-06	4.39E-04	8.71E-07
Pyrene	1.51E-04	5.71E-05	6.25E-03	2.54E-05
1,3-Dinitrobenzene	NA	NA	NA	NA
2,4-Dinitrotoluene	1.81E-04	1.16E-05	7.49E-03	5.16E-06
2,6-Dinitrotoluene	1.81E-04	1.16E-05	7.49E-03	5.16E-06
Nitrobenzene	1.50E-05	7.65E-06	6.21E-04	3.40E-06
Pentachlorobenzene	NA	NA	NA	NA
1,2-Dichlorobenzene	1.51E-04	2.69E-05	6.25E-03	1.20E-05
1,4-Dichlorobenzene	1.81E-05	4.92E-06	7.49E-04	2.19E-06
Hexachlorobenzene	1.81E-04	1.15E-05	7.49E-03	5.11E-06
Pentachlorophenol	1.97E-04	6.68E-05	8.15E-03	2.97E-05
1,2,4-Trichlorobenzene	3.18E-05	6.45E-06	1.32E-03	2.87E-06
2,4,5-Trichlorophenol	4.69E-04	2.95E-05	1.94E-02	1.31E-05
2,4,6-Trichlorophenol	1.97E-05	1.97E-05	8.15E-04	8.75E-06
o-Naphthylamine	3.33E-04	1.34E-04	1.38E-02	5.95E-05
4-Nitroaniline	7.26E-05	7.26E-05	3.00E-03	3.23E-05
2-Nitroaniline	4.69E-04	2.83E-05	1.94E-02	1.26E-05
2,3,7,8-TCDD TEQ (Dioxin)	3.00E-08	3.00E-08	1.24E-06	1.33E-08

	1-Hour	30-Day
MAXIMUM NORMALIZED CHI/Q, [(ug/m³)/(g/s)]	41.39	0.444

BASIS:
ISCST3 modeling of Winter Season (December-February) using On-Site meteorological data, fixed climatological mixing height, and 3.2-km radius polar receptor grid used by MRI.

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Table 4-5

**Comparison of Potential Incinerator Trial-Burn Impacts
with National Ambient Air Quality Standards (NAAQS)
Drake Chemical Site**

Pollutant	Period	Maximum Impact ($\mu g/m^3$)	NAAQS ($\mu g/m^3$)
PM_{10}	24-Hour	3.0	150
Lead	Quarterly	0.04	1.5
Carbon Monoxide	1-Hour	113	40,000
	8-Hour	23[a]	10,000
Nitrogen Oxides	Annual	5.9	100

(a) Quarterly average used to approximate annual average concentration.

The locations of the maximum estimated concentrations are presented in Figures 4-2 through 4-5 for the 1 hr, 24 hr, 30 day, and quarterly averaging periods, respectively. Figures 4-2 through 4-5 show contour plots of the impacts in concentration (ug/m^3) per unit of emissions (1 g/s), superimposed on the local topography and geographical features. The elevation contours are based on the actual model grid elevation data, and the base map is produced using MapInfo® desktop mapping software's StreetInfo (1992) maps for Clinton County, Pennsylvania. The maximum estimated impact locations are indicated by red triangles. As shown in the figures, the maximum estimated impacts from the incinerator occur along the southern valley walls in the southeast quadrant of the model grid.

FIGURE 4-2

1-HOUR MAXIMUM UNITY CONCENTRATIONS
WINTER SEASON (DECEMBER-FEBRUARY)
ON-SITE METEOROLOGICAL DATA

MAXIMUM
IMPACT
RECEPTOR

SCALE 1" = 915 METERS

INCINERATOR
(RFW APPROX.)

RADIUS OF AREA (3.2 KM)
MODELED BY MRI

Dunnstown

Lock Haven

Flemington

Mill Hall

24-HOUR MAXIMUM UNITY CONCENTRATIONS WINTER SEASON (DECEMBER-FEBRUARY) ON-SITE METEOROLOGICAL DATA

FIGURE 4-3

MAXIMUM IMPACT RECEPTOR

SCALE 1" = 915 METERS

RADIUS OF AREA (3.2 KM) MODELED BY MRI

INCINERATOR (RFW APPROX.)

4-15

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FIGURE 4-4

30-DAY MAXIMUM UNITY CONCENTRATIONS
WINTER SEASON (DECEMBER-FEBRUARY)
ON-SITE METEOROLOGICAL DATA

MAXIMUM
IMPACT
RECEPTOR

SCALE 1" = 915 METERS

RADIUS OF AREA (3.2 KM)
MODELED BY MRI

INCINERATOR
(RFW APPROX.)

4-16

FIGURE 4-5

90-DAY MAXIMUM UNITY CONCENTRATIONS
WINTER SEASON (DECEMBER-FEBRUARY)
ON-SITE METEOROLOGICAL DATA

MAXIMUM
IMPACT
RECEPTOR

SCALE 1" = 915 METERS

RADIUS OF AREA (3.2 KM)
MODELED BY MRI

INCINERATOR
(RFW APPROX.)

Lock Haven

Flemington

Dunnstown

Castanea

Mill Hall

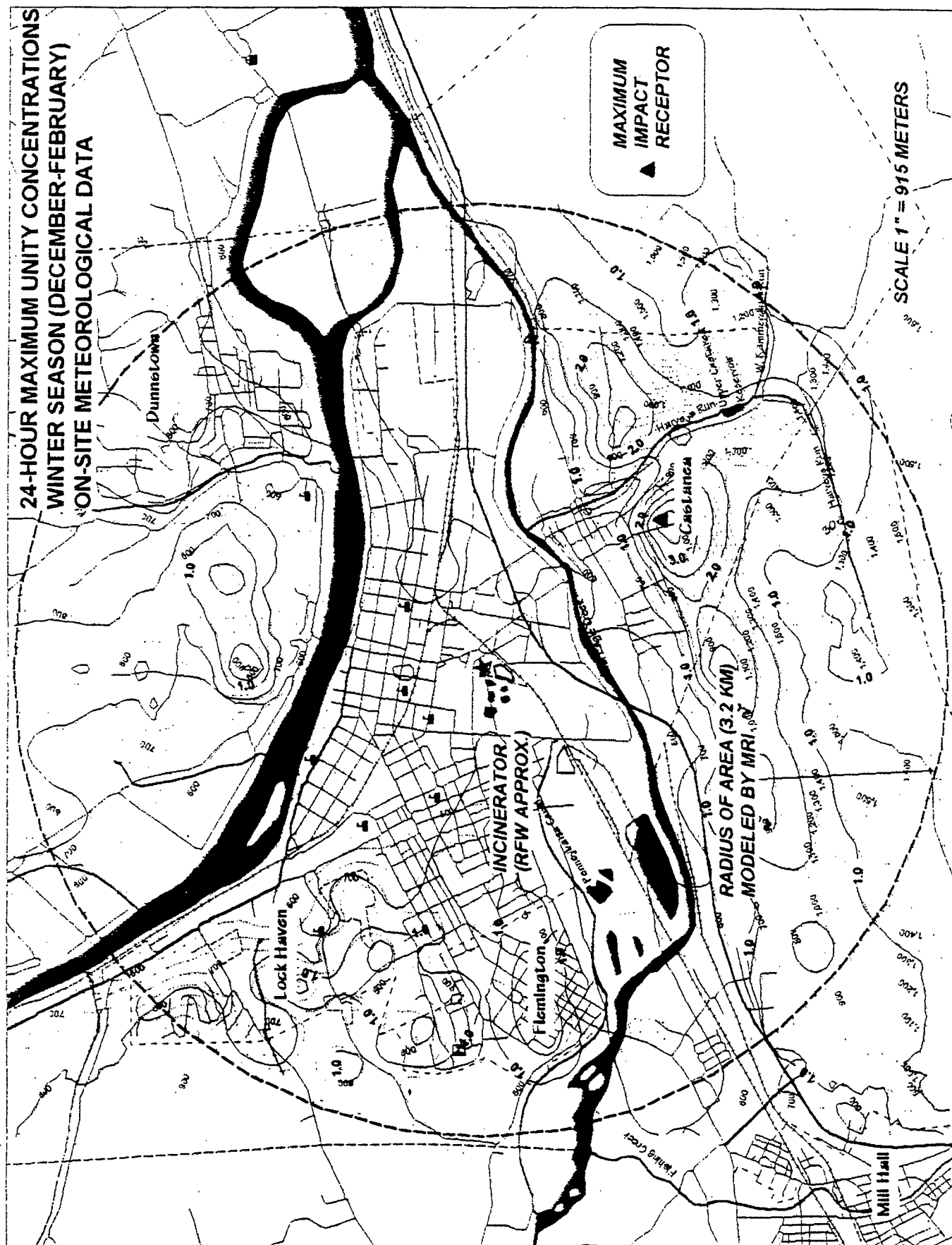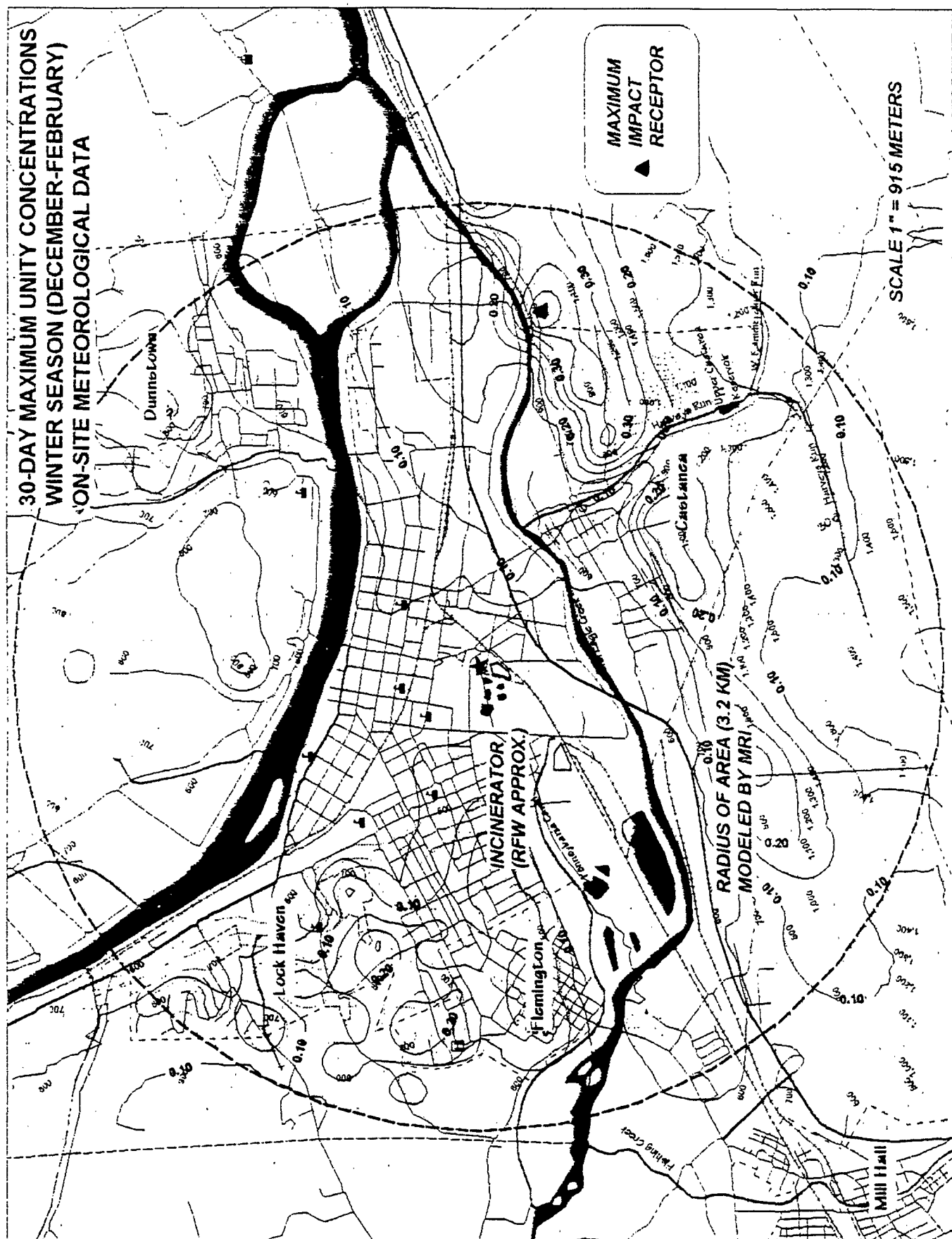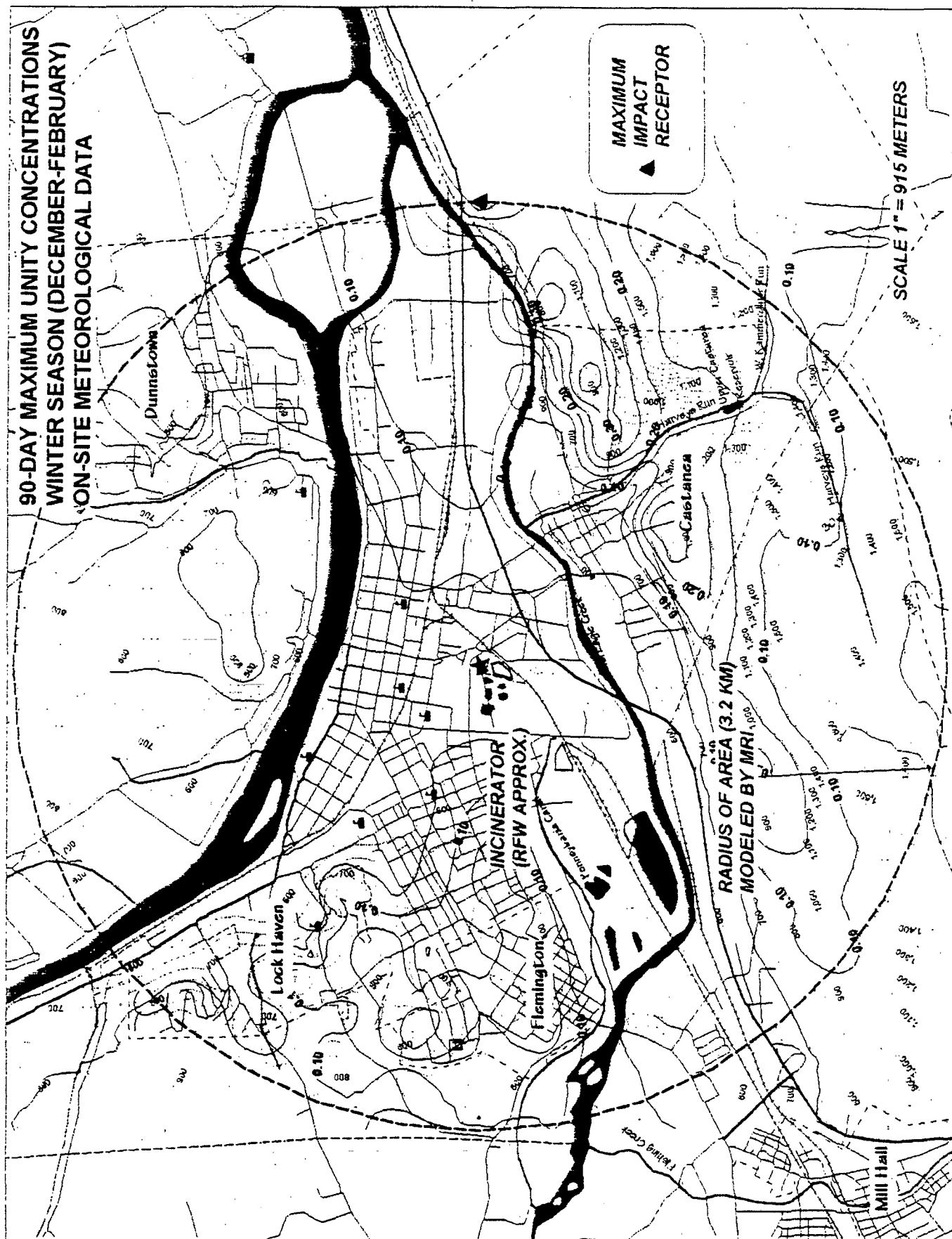AR308544

SECTION 5

HUMAN HEALTH RISK ASSESSMENT

5.1   INTRODUCTION AND APPROACH

This section of the report presents the process by which human health inhalation risks were evaluated from the predicted emissions from the Drake Chemical incinerator for the two-month trial burn period. A more detailed risk assessment will be performed in early 1996 for additional exposures to contaminants deposited on the ground and surface water in the vicinity of the Drake Chemical incinerator. Actual emissions data during the trial burn period will be used for the subsequent risk assessment, rather than predicted emissions.

A multistep process was used to estimate health risks by direct inhalation. Incinerator emissions during the two-month trial burn period were predicted from the waste stream characteristics (i.e., contaminated soil) based on the theoretical operation of the incinerator (Section 3). These emissions data were then evaluated by air dispersion modeling to predict short-term and long-term maximum air concentrations (Section 4). Carcinogenic and noncarcinogenic health risks were based on the 60-day duration of the proposed trial burn and were determined by comparing maximum predicted chemical air concentrations with appropriate long-term criteria.

The risk assessment process used very conservative assumptions in each of the steps. This ensured that risk was not underestimated. The impact of these conservative assumptions on risk is discussed in the uncertainty analysis.

5.2   DATA EVALUATION

5.2.1   Screening

Prior to quantitative inhalation risk evaluation, predicted air concentration of chemicals were compared with risk-based air concentrations (EPA, 1993). Risk-based concentrations (RBCs) are air levels associated with one-in-one million (1E-06) cancer risk or a hazard

quotient of 0.1. The inhalation RBCs were developed using conservative exposure assumptions and are considered protective of human health, i.e., are at risk levels that EPA would typically not take action to correct (EPA, 1991b).

To add conservatism to the screening process, the maximum predicted one hour air concentrations were compared to the chronic RBCs. The one hour concentrations represent maximum emission rates and the highest single concentration likely to occur on a short-term basis (i.e., average concentrations from the long-term operation of the incinerator will be lower). A chemical of potential concern (COPC) is a chemical whose maximum predicted one hour concentration exceeded the respective chronic RBC. This comparison ensures that a chemical will not be eliminated from the risk evaluation as a result of underestimation of its emission rate.

Table 5-1 presents these comparisons indicating which compounds were retained for further risk analysis. Several chemicals evaluated in the screening process are discussed below:

- Lead was retained as a COPC but could not be evaluated quantitatively because of the absence of chemical-specific toxicity criteria (these are discussed in the toxicity profiles). Lead was evaluated separately in Section 5.5.

- Nickel emitted by the incinerator was assumed to be the noncarcinogenic form. The carcinogenic forms of nickel (subsulfide, carbonyl, and refinery dust) are not associated with combustion processes like that proposed for the Drake Chemical incinerator because of the highly oxidizing conditions associated with the incineration process (EPA, 1991b).

5.2.2 Chemicals of Potential Concern

The COPCs selected from the screening process are summarized in Table 5-2. These COPCs were evaluated for subchronic inhalation risk as discussed in Subsection 5.5.

Table 5-1
Screening of Chemicals of Potential Concern [1]
Drake Chemical Incinerator

Chemical	Maximum 1-Hour Air Concentration (µg/m3)	Risk-Based Ambient Air Concentration (µg/m3)		Chemical of Potential Concern
Organics				
Benzene	1.88E-07	5.20E-02	(N)	No
2-Butanone	8.77E-05	1.00E+02	(N)	No
Carbon tetrachloride	1.06E-03	1.20E-01	(C)	No
Chlorobenzene	1.26E-03	2.10E+00	(N)	No
Chloroform	4.93E-06	7.80E-02	(C)	No
1,2-Dichlorobenzene	6.25E-03	2.10E+01	(N)	No
1,4-Dichlorobenzene	7.49E-04	2.60E-01	(C)	No
1,1-Dichloroethene	1.06E-03	3.60E-02	(C)	No
1,2-Dichloroethane	1.55E-05	6.90E-02	(C)	No
2,4-Dinitrotoluene	7.49E-03	7.30E-01	(N)	No
2,6-Dinitrotoluene	7.49E-03	3.70E-01	(N)	No
Dioxins/furans (as 2,3,7,8-TCDD equivalents)	1.24E-06	4.00E-08	(C)	Yes
Hexachlorobenzene	7.49E-03	3.90E-03	(C)	Yes
beta-Naphthylamine	1.38E-02	NTV		Yes
4-Nitroaniline	3.00E-03	1.10E+00	(N)	No
2-Nitroaniline	1.94E-02	2.10E-02	(N)	No
Nitrobenzene	6.21E-04	2.10E-01	(N)	No
PAHs				
Benzo(a)anthracene	2.63E-03	1.00E-02	(C)	No
Benzo(b)fluoranthene	2.57E-03	1.00E-02	(C)	No
Benzo(k)fluoranthene	1.81E-03	1.00E-01	(C)	No
Benzo(a)pyrene	2.13E-03	1.00E-03	(C)	Yes
Chrysene	3.25E-03	1.00E+00	(C)	No
Dibenzo(a,h)anthracene	6.87E-04	1.00E-03	(C)	No
Indeno(1,2,3-cd)pryene	1.38E-03	1.00E-02	(C)	No
Naphthalene	4.39E-04	1.50E+01	(N)	No
Pyrene	6.25E-03	1.10E+01	(N)	No
Pentachlorophenol	8.15E-03	5.20E-02	(C)	No
1,1,2,2-Tetrachloroethane	1.06E-03	3.10E-02	(C)	No
Tetrachloroethylene	2.38E-06	3.10E+00	(C)	No
Toluene	4.93E-06	4.20E+01	(N)	No
1,2,4-Trichlorobenzene	1.32E-03	9.40E-01	(N)	No
1,1,1-Trichloroethane	2.69E-06	1.00E+02	(N)	No
1,1,2-Trichloroethane	1.06E-03	1.10E-01	(C)	No
Trichloroethylene	1.25E-07	1.00E+00	(C)	No
2,4,5-Trichlorophenol	1.94E-02	3.70E+01	(N)	No
2,4,6-Trichlorophenol	8.15E-04	5.70E-01	(C)	No
Vinyl chloride	1.06E-03	2.10E-02	(C)	No
Inorganics				
Arsenic	1.32E+00	4.10E-04	(C)	Yes
Barium	5.96E+00	5.20E-02	(N)	Yes
Beryllium	3.60E-03	7.50E-04	(C)	Yes
Cadmium	1.77E+01	9.90E-04	(C)	Yes
Chromium III	4.22E-01	2.10E-04	(N)	Yes
Chromium VI	4.22E-01	1.50E-04	(C)	Yes
Lead	7.33E+01	NTV		Yes
Mercury	5.63E+00	3.10E-02	(N)	Yes
Nickel	1.92E+00	7.30E+00	(N) [b]	No
Selenium	3.00E-01	1.80E+00	(N)	No
Silver	1.19E-01	1.80E+00	(N)	No
Acid gases				
Hydrogen chloride	2.09E+01	7.30E-01	(N)	Yes

[1] = Screening performed according to EPA Region III (EPA, 1993).

[b] = Represents the toxicity value for nickel soluble salts.

(C) = Carcinogenic risk-based concentration (cancer risk = 1E-06).

(N) = Noncarcinogenic risk-based concentration (HQ = 0.1).

NTV = No toxicity value available.

Table 5-2

Contaminants of Potential Concern
Drake Chemical Incinerator

Organic Compounds

Dioxins/Furans (as 2,3,7,8-TCDD equivalents)
Hexachlorobenzene
beta-Naphthylamine
Benzo[a]pyrene

Inorganic compounds

Arsenic
Barium
Beryllium
Cadmium
Chromium III
Chromium VI
Lead
Mercury

Acid Gas

Hydrogen chloride (HCl)

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5.3   EXPOSURE ASSESSMENT

5.3.1   Introduction

This risk assessment evaluated inhalation exposure to the maximum exposed individual (MEI) from predicted maximum daily incinerator emissions during the proposed two-month trial burn period. Inhalation exposure is expressed as a daily intake dose per unit body weight (mg/kg-d). The intake dose for each chemical is a function of the exposure concentration, the frequency and duration of exposure, and the body weight of the individual.

$$\frac{\text{Inhalation}}{\text{Intake dose}} = \frac{\begin{array}{c}\text{Exposure} \\ \text{Concentration} \times \end{array} \begin{array}{c}\text{Inhalation} \\ \text{Rate} \times \end{array} \begin{array}{c}\text{Exposure} \\ \text{Duration} \times \end{array} \begin{array}{c}\text{Exposure} \\ \text{Frequency} \end{array}}{\text{BodyWeight(kg)} \times \text{Averaging time(days)}}$$

$$\text{(mg/kg-d)} \qquad (mg/m^3) \qquad (m^3/d) \qquad (yrs) \qquad (days/yr)$$

The intake doses were adjusted for carcinogenic and noncarcinogenic effects as discussed in Subsection 5.3.3. The purpose of expressing exposure in units of mg/kg-d is so that they can be related directly to toxicity criteria (also in units of mg/kg-d) for the calculation of health risks. The process of estimating health risk from chemical exposure and toxicity information is discussed in Subsection 5.5.

5.3.2   Subchronic Inhalation Exposures for the Child and Adult

Continuous subchronic exposure was evaluated during the entire two-month trial burn period separately for the child and adult. Subchronic exposures, as defined by EPA (1989a) are less than seven years in duration. Subchronic exposures were estimated from the maximum daily concentrations predicted from 30-day averages. The maximum 30-day value for each chemical was assumed to be breathed continuously by the MEI for the two-month trial burn period. The 30-day maximum air concentration for any chemical is more

conservative (i.e., higher) than a daily value obtained from a 60-day average. This approach ensured that inhalation exposure over the 60-day trial burn was not underestimated.

Inhalation exposure doses for the child and adult were determined individually and expressed as a daily intake per unit body weight (mg/kg-d). The child and adult doses depend on physiological and anatomical characteristics of each age group. These exposure factors are represented as values in the upper end of the range of typical population values (i.e., they are conservative) and are chosen as not to underestimate exposure (EPA, 1989a; EPA, 1991a). The following exposure factors were used:

- Breathing rate
    - Child - $15m^3/d$
    - Adult - $20m^3/d$

- Body Weight
    - Child - 15 kg
    - Adult - 70 kg

Other important factors required for calculating the exposure dose are frequency and duration of exposure. It was assumed in this risk assessment that both the child and adult were continously exposed to the maximum 30 day air concentration of each chemical 24 hours per day for a total of 60 days.

5.3.3 Calculation of Inhalation Exposure Dose

Table 5-3 presents the general equation by which exposure doses to the child and adult were calculated for carcinogenic effects and noncarcinogenic effects, respectively. Carcinogenic and noncarcinogenic exposure doses were calculated differently because they must be consistent with the manner in which carcinogenic and noncarcingenic toxicity criteria are presented (EPA, 1989a). Carcinogenic toxicity criteria are expressed as an average lifetime daily dose. Therefore, the exposure dose calculated for the two-month trial burn must be averaged over a 70 year lifetime. Exposures to the maximum air concentrations were adjusted to reflect the two-month trial burn duration over a lifetime exposure duration of

Table 5-3

Model for Calculating Child and Adult Intakes
Through Inhalation
Drake Chemical Incinerator

$$\text{Intake from Inhalation (mg/kg-day)} = \frac{CA \times IR \times CF \times EF \times ED}{BW \times AT}$$

Where:

CA	=	Chemical concentration in air (ug/m^3)
IR	=	Inhalation rate (m^3/day)
CF	=	Conversion factor (10^{-3} mg/ug)
EF	=	Exposure frequency (days/year)
ED	=	Exposure duration (years)
BW	=	Body weight (kg)
AT	=	Averaging time (days)

Exposure Assumptions (child and adult residents):

CA	=	Maximum 30-day air concentration for each chemical evaluated.
IR	=	Child - 15 m^3/day (EPA, 1991a).
	=	Adult - 20 m^3/day (EPA, 1991a).
EF	=	350 days/year for the child and adult residents (EPA, 1991a).
ED	=	Child and adult - 0.17 years (2months/12 months per year).
BW	=	Child - 15 kg (EPA, 1991a).
	=	Adult - 70 kg (EPA, 1991a).
AT	=	Noncarcinogenic dose - Exposure duration (0.17 years) x 365 days/year (EPA, 1989a).
	=	Carcinogenic dose - 70 years x 365 days/year (EPA, 1989a).

840 months (i.e., 70 years = 840 months). In effect, this means that the total dose received over a two month exposure period was averaged over a 70-year duration to estimate the exposure to carcinogens (EPA, 1989a). Noncarcinogenic toxicity criteria are expressed as the highest dose to which a person can be exposed every day for a lifetime without developing any adverse effects. Therefore, noncarcinogenic exposure doses are calculated based on the maximum daily exposure likely. The specific exposure parameters used are summarized in Table 5-3.

5.3.4 Inhalation Exposure Doses for the Child and Adult Residents

Table 5-4 presents the carcinogenic and noncarcinogenic doses for the child and adult for each chemical predicted to be emitted by the Drake Chemical Incinerator. These doses were used with the chemical-specific toxicity criteria identified in Subsection 5.4 to estimate health risks.

5.4 TOXICITY ASSESSMENT

5.4.1 General

The toxicity assessment identifies and describes the toxicity criteria for the COPCs predicted to be emitted by the incinerator. Toxicity criteria are measures of the potency of carcinogens or noncarcinogens in producing their toxic effects.

In evaluating potential health risk, both carcinogenic and noncarcinogenic health effects must be considered. Excessive exposure to all pollutants can potentially produce noncarcinogenic health effects, while the potential for carcinogenic effects is limited to exposure to those substances classified by EPA as carcinogens. Therefore, carcinogenic toxicity criteria were identified for those chemicals classified as carcinogens, and noncarcinogenic criteria were identified for all pollutants of concern. Criteria are expressed in units of dose per weight (milligrams per kilogram per day; mg/kg-d).

Table 5-4
Cancer and Noncancer Doses for the Child and Adult through the Inhalation Pathway Drake Chemical Incinerator

Chemical	Maximum 30-Day Air Concentration ($\mu g/m^3$)	Child Inhalation Doses		Adult Inhalation Doses	
		Cancer Dose (mg/kg-day)	Noncancer Dose (mg/kg-day)	Cancer Dose (mg/kg-day)	Noncancer Dose (mg/kg-day)
Organics					
Dioxins/furans (as 2,3,7,8-TCDD equivalents)	1.33E-08	3.10E-14	1.28E-11	8.85E-15	3.64E-12
Hexachlorobenzene	5.11E-06	1.19E-11	4.90E-09	3.40E-12	1.40E-09
beta-Naphthylamine	5.95E-05	1.39E-10	5.71E-08	3.96E-11	1.63E-08
PAHs					
Benzo(a)pyrene	9.15E-06	2.13E-11	8.77E-09	6.09E-12	2.51E-09
Inorganics					
Arsenic	4.93E-03	1.15E-08	4.73E-06	3.28E-09	1.35E-06
Barium	1.88E-02	NA	1.80E-05	NA	5.15E-06
Beryllium	1.85E-05	4.31E-11	1.77E-08	1.23E-11	5.07E-09
Cadmium	2.28E-02	5.31E-08	2.19E-05	1.52E-08	6.25E-06
Chromium III	8.31E-04	NA	7.97E-07	NA	2.28E-07
Chromium VI	8.31E-04	1.94E-09	7.97E-07	5.53E-10	2.28E-07
Lead	4.24E-02	9.87E-08	4.07E-05	2.82E-08	1.16E-05
Mercury	8.53E-03	NA	8.18E-06	NA	2.34E-06
Acid gases					
Hydrogen chloride	2.24E-01	NA	2.15E-04	NA	6.14E-05

NA = Not applicable. Chemical is not classified as a carcinogen.

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The objectives of this section are to present the general approach to determining inhalation toxicity values, and to identify the chemical-specific values for each chemical.

5.4.2 Carcinogens

Chemical carcinogens are evaluated by EPA first by determining their weight-of-evidence for causing cancer, and then estimating their cancer-causing potency expressed as a cancer slope factor (CSF). The weight-of-evidence classifications determined by EPA are A, B1, B2, C, D and E and are presented in Table 5-5. A, B, and C carcinogens for which cancer toxicity values were available are evaluated quantitatively in the human health risk assessment process (EPA, 1989a). The CSF for a chemical carcinogen quantitatively defines the relationship between exposure dose and carcinogenic response for a given chemical, and is the plausible upper-bound estimate of the probability of a response per unit intake of a chemical during an average lifetime exposure (70 years). CSFs are expressed as risk per unit dose in units of $(mg/kg\text{-}day)^{-1}$.

Human CSFs are usually derived from animal carcinogenicity studies. The models used to perform this extrapolation include assumptions that ensure the cancer risk to humans is not underestimated, and represent an upper-bound estimate of the likelihood of cancer at a given dose. When multiplied by the daily exposure dose estimated for the chemical at the site, cancer risk can be estimated. Because upper bound estimates of carcinogenic potency in humans are used, the actual (absolute) risk of cancer is unknown, but is likely to be considerably lower than the predicted (relative) risk, and may even be as low as zero (EPA, 1989a).

5.4.2.1 Inhalation Carcinogenic Criteria

Inhalation CSFs were used to evaluate the risk from exposure to potential carcinogens through the inhalation pathway at the Drake Chemical Site (Table 5-6). The EPA weight-of-evidence classifications for the COPCs are also provided. Values and classifications were obtained from the *Integrated Risk Information System* (IRIS; EPA, 1995a) or the *Health*

Table 5-5
EPA Categorization of Carcinogens
Based on Human and Animal Evidence

Human Evidence	EPA Categorization of Carcinogens (EPA, 1986)				
	Animal Evidence				
	Sufficient	Limited	Inadequate	No Data	No Evidence
Sufficient	A	A	A	A	A
Limited	B1	B1	B1	B1	B1
Inadequate	B2	C	D	D	D
No Data	B2	C	D	D	E
No Evidence	B2	C	D	D	E

Key:

Group A   Human carcinogen (sufficient evidence from epidemiological studies).

Group B1   Probable human carcinogen (at least limited evidence of carcinogenicity to humans).

Group B2   Probable human carcinogen (a combination of sufficient evidence in animals and inadequate data in humans).

Group C   Possible human carcinogen (limited evidence in animals in the absence of human data).

Group D   Not classified (inadequate animal and human data).

Group E   No evidence for carcinogenicity (no evidence for carcinogenicity in at least two adequate animals tests in different species, or in both epidemiological and animal studies).

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Table 5-6

Carcinogenic Slope Factors for Chemicals of Potential Concern

Drake Chemical Incinerator

Chemical	EPA Carcinogenicity Classification	Inhalation Cancer Slope Factor (mg/kg-day)[1]	Oral Cancer Slope Factor (mg/kg-day)[1]	Selected Cancer Slope Factor (mg/kg-day)[1]	Reference
Organics					
Dioxins/furans (as 2,3,7,8-TCDD equivalents)	B2	1.50E+05	1.56E+05	1.50E+05	EPA (1995b)
Hexachlorobenzene	B2	1.61E+00	1.60E+00	1.61E+00	EPA (1995a)
beta-Naphthylamine	B1	NTV	1.30E+02	1.30E+02 a	EPA (1993)
PAHs					
Benzo(a)pyrene	B2	NTV	7.30E+00	7.30E+00 a	EPA (1995a)
Inorganics					
Arsenic	A	1.51E+01	1.50E+00	1.51E+01	EPA (1995a)
Barium	--	NA	NA	NA	--
Beryllium	B2	8.40E+00	4.30E+00	8.40E+00	EPA (1995a)
Cadmium	B1	6.30E+00	NTV	6.30E+00	EPA (1995a)
Chromium III	--	NA	NA	NA	--
Chromium VI	A	4.20E+01	NTV	4.20E+01	EPA (1995a)
Lead	B2	NTV	NTV	NTV	--
Mercury	D	NA	NA	NA	--
Acid gases					
Hydrogen chloride	--	NA	NA	NA	--

a = Oral slope factor used in absence of inhalation slope factor.

NA = Not applicable. Chemical is not classified as a carcinogen.

NTV = No toxicity value available.

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Effects Assessment Summary Tables (HEAST; EPA, 1995b). If no inhalation CSFs were available, an oral CSF was used (i.e., route-to-route extrapolate). This is a conservative assumption because an inhalation carcinogen may not produce cancer by oral exposure. If no inhalation or oral carcinogencity criteria were avaiable for any substance classified as carcinogen, it was not evaluated quantitatively.

Several important observations about specific carcinogens are noted below:

- The inhalation slope factor for benzo[a]pyrene has been withdrawn by EPA (1995a). The oral slope factor was used as a default.

- Dioxins are a family of structurally-related chemicals (congeners) consisting of chlorinated dibenzo-p-dioxins and dibenzofurans. These congeners differ in the number and position of chlorine moieties on the parent chemical. The dioxins with carcinogenic potential are those with chlorines in the 2,3,7 and 8 positions and they differ in their cancer-causing potency. The dioxin emissions predicted in this risk assessment (Section 3) were modeled on the basis of all 2,3,7,8-congeners. To estimate risk, the resultant modeled air concentrations were multiplied by the CSF for 2,3,7,8-tetrachloro-dibenzobenzo-p-dioxin (TCDD; EPA, 1995b). Because 2,3,7,8-TCDD is the most potent carcinogen of the family, it is believed that this approach will result in a significant overestimation of risk from all dioxins.

- Although lead is classified as a B2 carcinogen, EPA has not developed a CSF with which to quantitate its potency (EPA, 1995a; EPA, 1995b). Lead was evaluated in the blood lead model for children discussed in the Risk Characterization Section (Subsection 5.5.3.2).

5.4.3 Noncarcinogenic Effects

5.4.3.1 Estimates of Noncarcinogenic Potency

Toxicity criteria used to evaluate potential noncarcinogenic health effects are termed reference doses (RfDs). Unlike the approach used in evaluating carcinogenic risk, it is assumed for noncarcinogenic effects that a daily threshold dose exists below which there is no potential for human noncancerous toxic effects. The RfD is defined as the daily dose to which a person can be continuously exposed without risk of appreciable deleterious

noncarcinogenic health effects (e.g., organ damage, biochemical alterations, birth defects; EPA, 1989a).

RfDs are developed for subchronic (2 weeks to 7 years) and chronic (greater than 7 years) exposure periods. The RfD is derived from toxicity-dose data obtained from human epidemiological studies or animal toxicity studies (EPA, 1988). It is important to note that the RfD is based on the premise that the measured toxic endpoint represents the most sensitive ("critical") target organ or tissue to the toxicity of that chemical (i.e., that target organ or tissue showing evidence of damage at the lowest dose). Since many chemicals can produce toxic effects on several organ systems, the distinction of the critical toxic effect provides added confidence that the RfD for a chemical represents the threshold dose below which no toxic effects on any organ system are likely to occur. Also, when the toxicity data are extrapolated to human protective levels, uncertainty factors are incorporated taking into account this extrapolation and the sensitivity of child and elderly populations.

5.4.3.2 Approach to Developing Inhalation Toxicity Criteria

Toxicity criteria for inhaled noncarcinogens can be expressed as the RfD (mg/kg-d) or reference concentration (RfC; mg/m^3). The RfD and RfC are interchangeable using standard conversion factors (EPA, 1995b). This conversion assumes that an adult weighs 70 kg and breathes 20m^3/day of air. In this report, all noncarcinogenic inhalation criteria were expressed as an RfD.

Subchronic and Chronic Inhalation Reference Doses

In humans, the term "chronic exposure" refers to continuous or intermittent exposure over a long duration (at least seven years, and usually a lifetime; EPA, 1989a). Chronic inhalation exposure limits are set low to minimize accumulation and toxicity of the chemical in the body over long periods of time. The term "subchronic exposure" refers to a shorter duration of exposure, usually on the order of weeks to months, and no more than seven years (EPA, 1989a). EPA has developed subchronic and chronic toxicity criteria for many

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chemicals. Because the proposed duration of the Drake Chemical incinerator trial burn is two months, the resulting inhalation exposure is most appropriately termed subchronic. Therefore, inhalation toxicity criteria that best approximated subchronic exposures were preferentially used. If no subchronic inhalation RfD was available, the chronic inhalation reference dose was used. Where chronic inhalation criteria were used as a default value, the potential for noncarcinogenic effects may have been overestimated since subchronic toxicity criteria are sometimes higher (less stringent) than chronic values. If inhalation criteria were not available, chronic oral RfDs were used as a default (i.e.; route-to-route extrapolation) as directed by EPA Region III (EPA, 1995d).

In summary, the hierarchy listed below was used to identify inhalation criteria.

1. Subchronic Inhalation RfD
2. Chronic Inhalation RfD
3. Chronic Oral RfD

Several chemicals evaluated for noncarcinogenic effects are discussed below:

- There was no inhalation or oral RfD for benzo[a]pyrene. The oral RfD for pyrene was substituted as a default value.

- A provisional subchronic inhalation RfD was available for barium in HEAST (EPA, 1995b) and this was used in place of the chronic RfD.

- The inhalation RfD for cadmium was a provisional value developed by the EPA-National Center for Environmental Assessment (EPA-NCEA; formerly the Environmental Criteria Assessment Office or ECAO). This value was obtained from EPA Region III (EPA, 1995c).

- Chronic oral RfDs were used as inhalation RfDs for the following chemicals: hexachlorobenzene; benzo[a]pyrene; arsenic; cadmium; chromium III; and chromium VI.

- Lead could not be evaluated quantitatively for noncarcinogenic effects because no RfD has been approved by EPA (1995a). Lead was evaluated by estimating childhood blood lead levels (see Subsection 5.5.3.2; EPA, 1994d).

- The inhalation RfD for mercury was a provisional value obtained from HEAST (EPA, 1995b).

- There is not a currently approved RfD for dioxins. Therefore, it could not be quantitatively evaluated.

Table 5-7 presents the inhalation criteria identified for noncarcinogenic effects, and the sources of information from which they were obtained.

5.5   RISK CHARACTERIZATION

5.5.1   Introduction

Chemical risk is a function of its exposure dose and its potential for toxicity. Therefore, the information presented in Sections 5.3 (Exposure Assessment) and 5.4 (Toxicity Assessment) were integrated as discussed below to evaluate carcinogenic and noncarcinogenic risk.

5.5.2   Carcinogenic Risk

Inhalation carcinogenic risk for each chemical was obtained by multiplying the chemical-specific CSF by the respective maximum 30-day average value (EPA, 1989a). The maximum impact concentration was adjusted in the calculation, as previously discussed, for the duration of exposure by additionally multiplying by a factor of 2/840 months (i.e., 2 months duration of exposure averaged over 70 years or 840 months). The probability of developing cancer is expressed in risk assessments as lifetime excess cancer risk (e.g., one excess cancer case occurring in one million exposed persons). This concept assumes that the risk of cancer from the given chemical is in "excess" of the background risk of developing cancer (i.e., approximately 1 in 3 chances during a lifetime, according to the American Cancer Society). Chemical specific cancer risks were then summed to estimate total cancer risk. In the context of this risk assessment, total cancer risk would be equivalent to the probability of developing cancer over a lifetime if exposed by inhalation for two months to all the carcinogens emitted by the incinerator. Cancer risk was estimated separately for the child and the adult.

Table 5-7
Noncarcinogenic Reference Doses for Chemicals of Potential Concern
Drake Chemical Incinerator

Chemical	Inhalation RfD (mg/kg-day)	Oral RfD (mg/kg-day)	Selected Toxicity Criterion (mg/kg-day)	Source of Criterion
Organics				
Dioxins/furans (as 2,3,7,8-TCDD equivalents)	NTV	NTV	NTV	--
Hexachlorobenzene	NTV	8.00E-04	8.00E-04	EPA (1995a)
beta-Naphthylamine	NTV	NTV	NTV	--
PAHs				
Benzo(a)pyrene	NTV	3.00E-01 [a]	3.00E-01	EPA (1995b)
Inorganics				
Arsenic	NTV	3.00E-04	3.00E-04	EPA (1995a)
Barium	1.43E-03 [b]	7.00E-02	1.43E-03	EPA (1995b)
Beryllium	NTV	5.00E-03	5.00E-03	EPA (1995a)
Cadmium	5.71E-05 [c]	5.00E-04	5.71E-05	NCEA (EPA, 1995c)
Chromium III	NTV	1.00E+00	1.00E+00	EPA (1995a)
Chromium VI	NTV	5.00E-03	5.00E-03	EPA (1995a)
Lead	NTV	NTV	NTV	--
Mercury	8.57E-05	3.00E-04	8.57E-05	EPA (1995b)
Acid gases				
Hydrogen chloride	5.71E-03	NTV	5.71E-03	EPA (1995a)

a The oral RfD for pyrene (EPA, 1995) was used for benzo(a)pyrene.

b The inhalation RfD for barium was converted from the subchronic RfC (5.00E-03 mg/m3) reported in HEAST (EPA, 1995b).

c The inhalation RfD for cadmium was a provisional value developed by EPA-NCEA regional support division as reported in EPA (1995c).

NTV = No toxicity value available.

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Lifetime incremental cancer risks estimated through the inhalation pathway are summarized in Table 5-8. Total lifetime cancer risk for the Drake Chemical incinerator was 6E-07 for the child and 2E-07 for the adult. The highest cancer risks for both child and adult were associated with the inhalation of cadmium and chromium VI. The total cancer risks are well below typical levels (i.e., 1E-06) of regulatory concern (EPA, 1991c).

5.5.3 Noncarcinogenic Effects

Potential for noncarcinogenic risk for each chemical was determined by dividing the child and adult chemical-specific exposure doses with their respective RfDs. This ratio for each chemical is referred to as the hazard quotient (HQ). If the HQ exceeds one, there is potential for noncarcinogenic effects from that chemical. If the HQ is less than one, the risk from exposure to that chemical is considered insignificant and below levels of regulatory concern (EPA, 1991c).

Chemical-specific HQs were summed for the child and the adult resulting in a measure of total noncarcinogenic potential referred to as the hazard index (HI). If the HI is less than one, there is likely no potential for noncarcinogenic effects from all chemicals evaluated. If the HI exceeds one, there is potential health concern.

5.5.3.1 Subchronic Noncarcinogenic Effects

HQs were calculated for the child and adult for each chemical by dividing the dose resulting from the maximum 30-day ambient concentration by the respective chemical-specific RfD. The child and adult HIs were determined by summing the respective chemical-specific HQs. Table 5-9 presents the subchronic HQs and HIs. All of the chemicals had HQs less than one for both the child and adult. The total HI for subchronic effects in the child was 0.54 and for the adult was 0.16. The greatest contributors to the HI for both the child and the adult were cadmium and mercury. The results indicate that noncarcinogenic effects

Table 5-8
Potential Cancer Risks for the Child and Adult
through the Inhalation Pathway
Drake Chemical Incinerator

Chemical	Child Inhalation Cancer Dose (mg/kg-day)	Adult Inhalation Cancer Dose (mg/kg-day)	Selected Cancer Slope Factor (mg/kg-day)[-1]	Potential Cancer Risk Child	Potential Cancer Risk Adult
Organics					
Dioxins/furans (as 2,3,7,8-TCDD equivalents)	3.10E-14	8.85E-15	1.50E+05	4.6E-09	1.3E-09
Hexachlorobenzene	1.19E-11	3.40E-12	1.61E+00	1.9E-11	5.5E-12
beta-Naphthylamine	1.39E-10	3.96E-11	1.30E+02 [a]	1.8E-08	5.1E-09
PAHs					
Benzo(a)pyrene	2.13E-11	6.09E-12	7.30E+00 [a]	1.6E-10	4.4E-11
Inorganics					
Arsenic	1.15E-08	3.28E-09	1.51E+01	1.7E-07	5.0E-08
Barium	NA	NA	NA	NA	NA
Beryllium	4.31E-11	1.23E-11	8.40E+00	3.6E-10	1.0E-10
Cadmium	5.31E-08	1.52E-08	6.30E+00	3.3E-07	9.6E-08
Chromium III	NA	NA	NA	NA	NA
Chromium VI	1.94E-09	5.53E-10	4.20E+01	8.1E-08	2.3E-08
Lead	9.87E-08	2.82E-08	NTV	NTV	NTV
Mercury	NA	NA	NA	NA	NA
Acid gases					
Hydrogen chloride	NA	NA	NA	NA	NA
			Total	6.1E-07	1.7E-07

[a] = Oral slope factor used in absence of inhalation slope factor.
NA = Not applicable. Chemical is not classified as a carcinogen.
NTV = No toxicity value available.

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Table 5-9
Hazard Indices for the Child and Adult through the Inhalation Pathway
Drake Chemical Incinerator

Chemical	Child Inhalation Noncancer Dose (mg/kg-day)	Adult Inhalation Noncancer Dose (mg/kg-day)	Selected Toxicity Criterion (mg/kg-day)	Hazard Index Child	Hazard Index Adult
Organics					
Dioxins/furans (as 2,3,7,8-TCDD equivalents)	1.28E-11	3.64E-12	NTV	NTV	NTV
Hexachlorobenzene	4.90E-09	1.40E-09	8.00E-04	6.1E-06	1.8E-06
beta-Naphthylamine	5.71E-08	1.63E-08	NTV	NTV	NTV
PAHs					
Benzo(a)pyrene	8.77E-09	2.51E-09	3.00E-01	2.9E-08	8.4E-09
Inorganics					
Arsenic	4.73E-06	1.35E-06	3.00E-04	1.6E-02	4.5E-03
Barium	1.80E-05	5.15E-06	1.43E-03	1.3E-02	3.6E-03
Beryllium	1.77E-08	5.07E-09	5.00E-03	3.5E-06	1.0E-06
Cadmium	2.19E-05	6.25E-06	5.71E-05	3.8E-01	1.1E-01
Chromium III	7.97E-07	2.28E-07	1.00E+00	8.0E-07	2.3E-07
Chromium VI	7.97E-07	2.28E-07	5.00E-03	1.6E-04	4.6E-05
Lead	4.07E-05	1.16E-05	NTV	NTV	NTV
Mercury	8.18E-06	2.34E-06	8.57E-05	9.5E-02	2.7E-02
Acid gases					
Hydrogen chloride	2.15E-04	6.14E-05	5.71E-03	3.8E-02	1.1E-02
			Total	5.4E-01	1.6E-01

NTV = No toxicity value available.

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associated with the operation of the incinerator are less than regulatory levels of concern (i.e., hazard index of 1) (EPA, 1991c).

5.5.3.2   Blood Lead Levels in Children

Cancer slope factors or reference doses have not been developed for lead. EPA believes that the available studies in animals and humans do not provide sufficient quantitative information for their calculation (EPA, 1995a; ATSDR, 1993). There is no scientific concensus as to the exact exposure dose of lead required to induce toxicity. However, lead toxicity studies in recent years have determined that the critical effect of concern for lead poisoning in the U.S. relates to neurotoxic effects in children (ATSDR, 1993). These effects, such as learning disabilities, hyperactivity and lowered IQ scores have been shown to be related to the blood lead levels in children. The Centers for Disease Control (CDC, 1991) currently believes that blood lead levels in children should not exceed 10 ug/dL in order to be protective of public health. The Integrated Exposure Uptake Biokinetic (IEUBK) model has been progressively developed by the EPA over the last 10 years. This model allows for the prediction of blood lead levels in children up to age seven based on knowledge of potential exposure concentrations (EPA, 1994d). The model predicts blood lead levels on the basis of scientific studies of how lead is absorbed, metabolized, distributed, and excreted by susceptible human populations.

The maximum 30-day air concentration of lead (0.042 ug/m^3) was substituted in the model. It was conservatively assumed that the child would be exposed to U.S. average background levels of lead received in the diet, by soil/dust ingestion, from maternal blood at birth, and drinking water consumption. Additionally, standard, conservative risk assessment exposure assumptions were incorporated, such as breathing rate. The results of the lead modeling yielded a mean blood value of 1.6 ug/dL which is less than one-sixth of the threshold level for adverse effects in young children. These results strongly support the view that blood lead levels in children will not be significantly affected by the two-month trial burn of the Drake Chemical incinerator.

5.6   UNCERTAINTY ANALYSIS

5.6.1   Introduction

The objectives of the uncertainty analysis are to present and discuss the assumptions used in the risk assessment with the greatest impacts on the risks. The goals of the analysis are specifically defined in two guidance documents provided by the EPA (1989a; 1992b):

- Provide to the appropriate decision makers a summary of those factors which significantly influence the risk results, evaluate their range of variability, and assess the contribution of these factors to the under- or overestimation of risk.

- Discuss the data underlying the assumptions that most significantly influence risk to highlight the strengths and weaknesses of the risk assessment results.

The predicted carcinogenic and noncarcinogenic risk estimates were based on a number of assumptions that incorporated varying degrees of uncertainty resulting from several sources, including those pertaining to:

- The estimation of emissions from the incinerator.

- The prediction of off-site concentrations.

- The exposure assessment.

- The toxicity assessment.

The following subsections describe these uncertainties in greater detail.

5.6.2   Uncertainty Associated with Emissions Estimates

Section 3 presented a detailed discussion of a number of assumptions that were made in estimating emissions. Some of the key uncertainties are included below:

- The maximum emission rate was based on the highest sampled soil concentration. The reality is that a composite of soils will be fed at an average concentration lower than the highest level. This would tend to overestimate the actual emissions and risk.

- Several compound emission rates were based on non-detect limits for the compounds. It is very likely that these levels are lower than the detection limit.

- Metals partitioning was based on a 1600°F temperature while the Drake incinerator will be operating at 1000-1200°F. For a number of the metals, this lower temperature will lead to lower emission rates than the emission estimates used in the risk assessment. This approach will tend to overestimate the risk associated with the metals.

- In cases where specific metal partition factors were not available, an assumption of 100% was used. This is an overestimate.

- Several metal concentrations exceeded 100 ppm. According to the OAQPS report (EPA, 1992a), partition factors reported in the document were likely to be overstated at metal concentrations greater than 100 ppm. This approach will also overestimate risk associated with the metals.

- Air pollution control device efficiencies are biased low in the OAQPS document (EPA, 1992a), which was used to estimate emission factors in the risk assessment. This will tend to overestimate risks.

The approaches taken in the estimation of emissions was conservative, i.e., to take certain approaches that were likely to overestimate predicted emission rates. While the degree of the overestimation will be uncertain prior to the performance of the trial burn, the approach to estimating emissions will likely result in an overestimate of predicted risks.

5.6.3 Uncertainty Associated with Air Dispersion Modeling

Section 4 presented the results of the dispersion modeling. Several assumptions were made that create uncertainties in the modeled air concentrations.

- In general, U.S. EPA air quality models have been designed to over-predict measured concentrations by a factor of 2-3. The over-prediction is applicable for areas of simple terrain (terrain below stack top elevation) and complex terrain (terrain above stack top elevation). Furthermore, each air quality model contains options that are designed to overestimate air concentrations. The use of U.S. EPA models and the regulatory default options ensures that conservative estimates of air concentrations are made.

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- The topography surrounding the Drake Chemical site has areas of complex terrain. High air quality concentrations are likely to occur at receptors where the terrain exceeds the height of the stack. Therefore, the selection of elevations for receptors is important. Rather than use the actual elevation at a receptor, the peak elevation surrounding the receptor was used. The selection of peak elevations rather than actual elevations will cause more conservative model predictions.

- The complex terrain algorithms (COMPLEX I) used in ISC3 represent a relatively simplified and conservative treatment of dispersion in complex terrain. More refined complex terrain dispersion algorithms are contained in the U.S. EPA Complex Terrain Dispersion Model Plus Algorithms for Unstable Situations (CTDMPLUS) model. However, The CTDMPLUS model requires meteorological data collected at multiple levels up to plume height. Since only one height level of on-site data was collected. further meteorological data would need to be collected at the site in order to utilize CTDMPLUS.

The approach used in modeling off-site concentrations was a standard EPA approach that has a certain level of overestimation built in. This level of conservatism is difficult to quantify, however, it is very likely to result in an overprediction of actual risks.

5.6.4 Uncertainty Associated with Exposure Assumptions

The exposure assumptions directly influence the amount of chemical predicted to accumulate in the individual. The concept of the maximally-exposed individual (MEI) was used to estimate. inhalation exposure potential for the individual. This approach was extremely conservative in that it assumed the same individual, whether child or adult, breathed the maximum predicted air concentration of all chemicals during the exposure period under evaluation. Average emission rates calculated in Section 3 were not used in the analysis. Moreover, the location of the various predicted maximum concentrations were at locations that are not generally populated. Although these areas are accessible to the public, it is not likely that an individual would be standing in this location for more than a few minutes during the course of the day, week, or month.

Again, the degree of conservatism and overprediction of risks that resulted from these assumptions are difficult to quantify; however it is very likely that the exposure assumptions alone would result in a significant overprediction of risk.

5.6.5 Uncertainties Associated with Toxicity Assumptions

The toxicological uncertainties relate to the methodology by which carcinogenic and noncarcinogenic criteria (i.e., cancer slope factors and reference doses) are developed. In general, the methodology currently used to develop cancer slope factors and reference doses is conservative, and likely results in overestimation of human toxicity (EPA, 1989a). Other uncertainties relate to factors such as route-to-route extrapolation of RfDs (i.e., oral to inhalation) and the use of chronic toxicity values as defaults for subchronic exposures.

5.6.5.1 Cancer Slope Factors

Although there is evidence to suggest some carcinogens may exhibit thresholds, CSFs are developed assuming there is no safe level of exposure to any chemical proven or suspected to cause cancer. This uncertainty implies that exposure to even a single molecule of a chemical may be associated with a finite risk, however small. The assumption is that even if relatively large doses of a pollutant were required to cause cancer in laboratory animals (i.e., much higher than a person would ever likely be exposed to over a lifetime), these exposure doses can be linearly extrapolated downward many orders of magnitude to estimate slope factors for humans. A significant uncertainty for the carcinogens is whether the cancer slope factors accurately reflect the carcinogenic potency of these chemicals at low exposure concentrations. The calculated slope factor is used to estimate an upperbound lifetime probability of an individual developing cancer as a result of exposure to a particular level of a carcinogen. Therefore, the cancer slope factors developed by EPA are generally conservative and represent the upperbound limit of the carcinogenic potency of each chemical. The actual risk posed by each chemical is unknown, but is likely to be lower than the calculated risk, and may even be as low as zero (EPA, 1989a). The conclusion is that these toxicity assumptions will result in an overestimation of carcinogenic risk.

5.6.5.2 Reference Doses

In the development of reference doses (RfDs), it is assumed by EPA (1989a) that a threshold dose exists below which there is no potential for adverse health effects to the most sensitive individuals in the population. The RfD is typically derived from dose-response studies in animals. The chemical NOAEL (no-observed-adverse-effect level) or the LOAEL (lowest-observed-adverse-effect level), assuming a NOAEL is unavailable, is divided by several uncertainty factors of 10 each and an optional modifying factor between 1 and 10 (EPA, 1988). The final degree of extrapolation for a given chemical usually ranges from 10 to 1,000 depending on the validity of the overall data and the confidence in the study. Therefore the RfD is usually one tenth to one-thousandth of the study dose (NOAEL or LOAEL). In general, the calculated RfD is overly protective, and its use will result in a moderate to high degree of overestimation of noncarcinogenic risk.

5.6.5.3 Use of Chronic RfDs for Subchronic Exposure

In a number of cases, chronic (greater than 7 year exposures) toxicity criteria were used to estimate subchronic effects. Chronic RfDs are developed assuming a lifetime daily exposure. Subchronic RfDs, which are usually based on an exposure duration of 2 weeks to 7 years, generally tend to be greater than chronic RfDs, and therefore, would result in a lower hazard quotient and index.

5.6.5.4   Chemicals With No Quantitative Toxicity Criteria

Several COPCs could not be evaluated quantitatively in this risk assessment because EPA has not developed cancer slope factors or reference doses:

- Dioxins/Furans - lacks noncancer RfDs
- beta-Naphthylamine - lacks noncancer RfD
- Lead - lacks cancer slope factor and noncancer RfD

Therefore, total cancer risks and noncarcinogenic hazard indices for the child and adult were underestimated.

5.6.5.5 Chemicals Eliminated As COPCs Based on Risk-Based Screening

A number of chemicals predicted to be present in the contaminated soil were eliminated from further risk evaluation based on the screening procedure used in Subsection 5.5.1. Because they are associated with a small risk, their elimination from the total risk estimates may have resulted in a slight underestimation. However, because of the conservative nature of the screening process, their cumulative impact on risk is likely to be low and would and is unlikely to contribute significantly to the total.

5.6.5.6 Route-To-Route Extrapolation

Carcinogenic risks and noncarcinogenic hazard indices may have been slightly under- or overestimated because of the substitution of oral toxicity criteria for inhalation effects when there were no inhalation values available. There is uncertainty as to the nature and degree of toxicity by the inhalation route of exposure from these chemicals. Route-to-route extrapolation was used for the following chemicals:

- Benzo[a]pyrene - cancer slope factor; noncancer RfD
- beta-Naphthylamine - cancer slope factor
- Hexachlorobenzene - noncancer RfD
- Arsenic - noncancer RfD
- Beryllium - noncancer RfD
- Chromium III - noncancer RfD
- Chromium VI - noncancer RfD

5.6.6 Summary of Uncertainties

The net effect of the various approaches used to estimate incinerator emissions, model off-site air concentrations, evaluate potential exposure, and incorporate available toxicity data is likely to result in an overprediction of risk. The cumulative effect of this conservatism is very likely

to result in a significant overestimation of potential health risks. Additional effort to describe the magnitude of the impact of these uncertainties was not included in this risk assessment because all the predicted risks were below levels of regulatory concern. Although there are some assumptions that may have underestimated risk, the contribution of these factors was small relative to the degree of overestimation in the overall risk assessment process. Table 5-10 summarizes these assumptions and uncertainties and indicates their impact on the over- or underestimation of risk.

Table 5-10

Summary of Uncertainty Analysis
Drake Chemical Incinerator

Uncertainty Element	Effects on Risk Estimate		
	Potential for Overestimation	Potential for Underestimation	Potential for Over- or Underestimation
Emissions/Air Modeling			
• Prediction of chemical emissions	High		
• Estimation of maximum air exposure concentrations	High		
Exposure Assessment			
• Exposure scenario locations	Moderate		
• Standard assumptions regarding inhalation rates, and life expectancy	Moderate-High		
Toxicity Assessment			
• Cancer slope factors	Moderate-High		
• Reference doses	Moderate-High		
• Use of chronic RfCs for subchronic	Low-Moderate		
• Route-to-route extrapolation			Low

SECTION 6

CONCLUSIONS

EPA (1992b) states that a total excess cancer risk less than 1E-06 (one excess cancer risk in 1,000,000 individuals) at a site does not require remediation, and is an acceptable risk from a public health standpoint. In addition, EPA has stated that cancer risks between 1E-06 and 1E-04 may be acceptable if site-specific conditions justify them (EPA, 1991c). In view of the conservative assumptions made in estimating chemical emissions, air concentrations, exposure, and toxicity, and in view of the fact that the estimated risk is below 1E-06 (6E-07, child; 2E-07, adult), it is concluded that the two-month trial burn at the Drake Chemical incinerator does not pose a significant cancer risk. Additionally, the hazard indices calculated for the subchronic child and adult exposures were less than one. EPA (1991c) indicates that total hazard indices less than one are unlikely to cause adverse health impacts. This indicates that no acute or subchronic health risks are expected from exposure to emissions from the incinerator during the two-month trial burn.

SECTION 7

REFERENCES

ATSDR (Agency for Toxic Substances and Disease Registry). 1993. *Toxicological Profile for Lead.* Final. NTIS PB 93-182475. April 1993.

Auer, A.H. 1978. *Correlation of Land Use and Cover with Meteorological Anomalies.* J. Appl. Meteorol. 17: 636-643.

CDC (Centers for Disease Control). 1991. *Preventing Lead Poisoning in Young Children - A Statement by the Centers for Disease Control.* October 1991.

EPA (U.S. Environmental Protection Agency). 1978. *National Primary and Secondary Ambient Air Quality Standards for Lead.* Fed. Reg. 43, No. 194, pp. 45978-46285, 5 October 1978.

EPA (U.S. Environmental Protection Agency). 1986. *Guidelines for Carcinogenic Risk Assessment.* Fed. Reg. Vol. 51, No. 185, 33992.

EPA (U.S. Environmental Protection Agency), 1988. *Reference Dose (RfD): Description and Use in Health Risk Assessments (Background Document I)*, Technical Information Center, D. Barnes, 23 March, 1988.

EPA (U.S. Environmental Protection Agency). 1989a. *Risk Assessment Guidance for Superfund: Human Health Evaluation Manual, Part A.* Interim Final. Office of Solid Waste and Emergency Response. OSWER Directive 9285.701A.

EPA (U.S. Environmental Protection Agency). 1989b. *Review of the National Ambient Air Quality Standards for Lead. Assessment of Scientific and Technical Information.* OAQPS Draft Staff Paper, Air Quality Management Division, Office of Air Quality Planning and Standards, Research Triangle Park, NC 27711, March 1989.

EPA (U.S. Environmental Protection Agency), 1991a. *Risk Assessment Guidance for Superfund, Volume I - Human Health Evaluation Manual Supplemental Guidance, "Standard Default Exposure Factors."* Interim Find. OSWER Directive 9285.6-03.

EPA (U.S. Environmental Protection Agency. 1991b. *Code of Federal Regulations, 40 CFR Parts 260, 261, 263, 265, 266, 270 and 271 (Burning of Hazardous Waste in Boilers and Industrial Furnaces; Final Rule).* Fed. Reg. Vol. 56, No. 35, February 21,1991.

EPA (U.S. Environmental Protection Agency), 1991c. *Role of the Baseline Risk Assessment in Superfund Remedy Selection Decisions.* Memorandum from Don R. Clay, Assistant Administrator.

EPA (U.S. Environmental Protection Agency). 1992a. *Screening Procedures for Estimating the Air Impacts of Incineration at Superfund Sites*, Air/Superfund National Technical Guidance Study Series, Report ASF-23. Office of Air Quality Planning and Standards (OAQPS), February 1992. PB92-171917.

EPA (U.S. Environmental Protection Agency). 1992b. *Guidance on Risk Characterization for Risk Managers and Risk Assessors*. Memorandum from F. Henry Habicht. II to Assistant and Regional Administrators. Feb. 26, 1992.

EPA (U.S. Environmental Protection Agency). 1993. *Selecting Exposure Routes and Contaminants of Concern by Risk-Based Screening*. EPA Region III Technical Guidance Manual, Risk Assessment. EPA/903/R-93-001. January 1993.

EPA (U.S. Environmental Protection Agency). 1994a. *Test Pit Analyses (Fixed Base level IV) for Organic Compounds, Table 4-5*. Drake Chemical Site-Phase III RI.

EPA (U.S. Environmental Protection Agency). 1994b. *Test Pit Analyses (Fixed Base) for Inorganic Analyses, Table 4-6*. Drake Chemical Site-Phase III RI.

EPA (U.S. Environmental Protection Agency). 1994c. *Guidelines on Air Quality Models (Revised)*. EPA Publication No. EPA-450/2-78-027R. Research Triangle Park, NC.

EPA (U.S. Environmental Protection Agency), 1994d. *Guidance Manual for the Integrated Exposure Uptake Biokinetic Model for Lead in Children*. EPA/540/R-93/081. PB93-963510. OERR, Washington, D.C. February, 1994.

EPA (U.S. Environmental Protection Agency). 1995a. *Integrated Risk Information System (IRIS), On-line Computer Database*. Environmental Criteria and Assessment Office, Cincinnati, Ohio.

EPA (U.S. Environmental Protection Agency. 1995b. *Health Effects Assessment Summary Tables*, FY-1995 Annual, OERR 9200.6-303 (95-1), NTIS No. PB95-921199, March 1995.

EPA (U.S. Environmental Protection Agency. 1995c. *EPA Region III Risk-Based Concentration Table*. U.S. EPA Region III, Philadelphia, PA 19107, October 20, 1995.

EPA (U.S. Environmental Protection Agency. 1995d. *Personal Communication between Nancy Rios (EPA, Region III) and Robert Warwick (WESTON)*. 17 November 1995.

Gross, G. 1995. Personal Communication between Gary Gross, U.S. EPA Region III, RCRA, with Paul Seibert, Roy F. Weston, Inc. November 30, 1995.

Holzworth, G.C. 1972. *Mixing heights, Wind Speeds, and Potential For Urban Air Pollution Throughout the United States*. Division of Meteorology, U.S. EPA, Office of Air Programs, Research Triangle Park, NC. January 1972.

MITRE (MITRE Corporation). 1983 *Hazardous Waste Stream Trace Metal Concentrations and Emissions*, U.S. EPA Office of Solid Waste, November 1983.

Montgomery Watson (1994). *Test Pit Excavation Report, Drake Chemical Superfund Site, Lock Haven, Pennsylvania.* October 1994.

Montgomery Watson. 1993. *Drake Chemical Superfund Site, Lock Haven, Pennsylvania, Six Month Meterological Monitoring Report.* Walnut Creek, CA. 1993.

MRI (Midwest Research Institute). 1988. Guidance on Carbon Monoxide Controls for Hazardous Waste Incinerators. Draft Final Report to U.S. EPA Office of Solid Waste. EPA Contract No. 68-01-7287. Work Assignment 27. September 9, 1988.

MRI (Midwest Research Institute). 1993. Drake Chemical Superfund Site, Lock Haven, Pennsylvania, Six-Month Meterological Report. Walnut Creek, CA.

MRI (Midwest Research Institute). 1994. *Trial Burn Plan for the Drake Chemical Superfund Site's Mobile Hazardous Waste Incinerator. Volume I. Trial Burn Plan.* For RUST International, Inc. Palos Heights, IL 60463. MRI Project No. 3620-01. October 24, 1994.

APPENDIX A

TOXICOLOGY PROFILES

ARSENIC

Inorganic arsenic is toxic by the inhalation and oral routes. Humans exposed to arsenic near hazardous waste sites could inhale arsenic dust in the air, ingest it in water, food, or soil, or contact it dermally in soil or water. The greatest effect via inhalation is the increased risk of lung cancer. Orally, the effects most likely to be seen are gastrointestinal irritation, nerve and blood problems, and a group of skin diseases, including cancer. The main effect of direct dermal contact with inorganic arsenic is local irritation and dermatitis.

Acute exposure to arsenic has caused death due to heart and lung failure, while death caused by repeated exposure has resulted from the failure of more than one tissue injured by arsenic. Inhalation of inorganic arsenic dusts (mainly arsenic trioxide) irritates the nasal passages. However, workers exposed to high levels of arsenic trioxide in air have usually shown no signs of chronic respiratory functional impairment. Injuries to the lung have been more pronounced following high (near lethal) oral doses. Orally, long-term exposure to low levels of inorganic arsenic has resulted in "Blackfoot disease" and gangrene. Both acute high dose and repeated low dose exposures can cause irritation of the gastrointestinal tract. Similar effects have been observed with subchronic or chronic inhalation exposure. Anemia and leukopenia are common observations in humans exposed to inorganic arsenic by the oral and inhalation routes. Kidney effects, largely vascular in origin, were found in humans orally exposed to inorganic arsenic. Skin lesions are an early sign of chronic oral exposure to inorganic arsenic. Certain lesions (i.e., hyperkeratinized corns) may develop into skin cancer.

Neurological effects are common in humans exposed orally to arsenic and have been reported in some workers exposed by inhalation. Acute high doses lead to brain dysfunction which can end in seizures and coma in more severe cases. Peripheral nerve damage has occured with lower-level exposure. Human data indicate that exposure to inorganic arsenic increases the chances of developing cancer. Lung cancer is the predominant effect by the inhalation route; some tumors have been observed at other sites. Increased skin cancer incidence has been observed in several populations consuming drinking water with high arsenic concentrations. Based on these findings the EPA has categorized arsenic in Group A, human carcinogen.

Reference

ATSDR (Agency for Toxic Substances and Disease Registry). 1993. Toxicological Profile for Arsenic. U.S. Department of Health and Human Services. Atlanta, GA. PB93-182376.

BARIUM

Some barium is found in water and food. As a result, it is readily available to people through these two media. Another pathway of exposure is by inhalation of low levels in ambient air. These exposures to barium should not pose a serious health hazard. Barium is also a regular contaminant at hazardous waste sites, therefore it may potentially cause health risks to humans living or working near the sites. Human evidence suggests that the cardiovascular system may be one of the main targets of acute barium toxicity. Other effects have been found to be caused by barium, but the lack of data or insufficient data does not allow conclusions to be drawn for some of them.

Human case studies have reported that acute oral ingestion of barium caused death. However, there were no deaths reported in humans following inhalation or dermal exposure. Case reports have shown that acute oral exposure to barium may result in respiratory deficiency and paralysis, heart and blood vessel effects, irritation of and bleeding in the stomach and the intestines, liver and renal deterioration, muscle weakness and paralysis, and neurological effects. Kidney failure and neurological effects were noted in individuals who inhaled an acute dose of barium. Human data have indicated that blood effects can result from acute inhalation, oral, and dermal exposures. Workers that were occupationally exposed to barium by the inhalation route developed benign lung problems. There have been no adequate human or animal studies that have evaluated the carcinogenic potential of barium. Barium has not been evaluated by EPA for its human carcinogenic potential.

Reference

ATSDR (Agency for Toxic Substances and Disease Registry). 1992. Toxicological Profile for Barium and Compounds. U.S. Department of Health and Human Services. Atlanta, GA. PB93-110658.

BERYLLIUM

Beryllium does not occur naturally in the earth's crust as a pure element, although it can be found as a chemical component of certain rocks, coal, oil, soil, and volcanic dust. Beryllium also occurs in some foods. People can be exposed to beryllium through air, food, water, and soil. The intake of beryllium for the general population is very low. Occupational exposure to beryllium is the primary route of human exposure to this chemical. Workers engaged in machining metals containing beryllium, in recycling beryllium from scrap alloys, or in using beryllium products can be exposed to higher levels of beryllium. The respiratory system and heart are the primary targets of toxicity in individuals exposed through the inhalation of beryllium.

In humans, death has resulted from respiratory distress caused by occupational exposure to beryllium. Human exposure to elevated concentrations of beryllium has resulted in beryllium pneumonitis with symptoms including cough, substernal burning, shortness of breath, anorexia, and increasing fatigue. Lower concentrations of the less soluble forms of beryllium have caused chronic beryllium lung disease characterized by granulomas, fibrosis, and emphysema. Beryllium exposure in the workplace has caused enlargement of the heart muscles. Some case reports showed hepatic and renal effects in workers, whereas a study of 25 people exposed to beryllium dust showed no liver effects during autopsies. A wide range of skin lesions and eye effects have resulted in humans exposed to beryllium in the workplace.

Epidemiology studies regarding beryllium's carcinogenic potential are considered to be inadequate. Beryllium has been shown to cause lung cancer through inhalation in rats and monkeys and to cause osteosarcomas in rabbits through intravenous or intramedullary injection. EPA has classified beryllium as a Group B2, probable human, carcinogen.

Reference

ATSDR (Agency for Toxic Substances and Disease Registry). 1993. Toxicological Profile for Beryllium. U.S. Department of Health and Human Services. Atlanta, GA. PB93-182392.

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CADMIUM

Cadmium is a metal that occurs naturally in all soils and rocks, and in coal and fertilizers. It is usually found in the environment as a mineral combined with other elements such as oxygen, chlorine, or sulfur. Cadmium is often found as a part of small particles present in the air. Fish, plants, and animals take up cadmium from the environment; therefore, humans can be exposed to cadmium through the ingestion of cadmium-contaminated food and water and through the inhalation of cadmium-contaminated air particles. Cadmium has no known beneficial effects on human health. Cadmium is a cumulative toxicant, and the exposure conditions of most concern are long-term exposure to elevated levels in the diet.

The inhalation of high levels of cadmium-contaminated air can severely damage the lungs, which may cause pulmonary edema. This is the primary cause of death following inhalation of cadmium, whereas fluid imbalance and widespread organ damage are reported in cases of death resulting from suicidal ingestion. The gastrointestinal tract is the target organ for acute oral exposure to cadmium, due to the direct irritation of the gastric epithelium. Anemia has occurred in humans following long-term oral and inhalation exposure to cadmium. Oral exposure to cadmium has been shown in animals to reduce the uptake of iron from the diet. The kidneys are the primary target organ of chronic oral and inhalation exposure to cadmium, with exposure leading to a build-up of cadmium in the kidneys that can cause damage to the renal tubules. Renal dysfunction following chronic inhalation or ingestion of cadmium could lead to painful debilitating bone disease in humans, particularly in individuals with malnutrition.

Genotoxic effects in humans following inhalation or oral exposure to cadmium are conflicting; however, there has been evidence that cadmium can alter chromosomes in mammalian cells. There is weak evidence that cadmium inhalation causes lung cancer in humans, but strong evidence exists that cadmium inhalation can cause lung cancer in rats. EPA has classified cadmium as a Group B1, probable human, carcinogen by inhalation.

Reference

ATSDR (Agency for Toxic Substances and Disease Registry). 1993. Toxicological Profile for Cadmium. U. S. Department of Health and Human Services. Atlanta, GA. PB93-182418.

CHROMIUM

Chromium is a naturally occurring element, found in the environment in several different forms. Of the available forms of chromium, those most commonly associated with industrial processes are chromium III and chromium VI. In industry, these forms of chromium are typically used for chrome plating, dye and pigment manufacturing, leather tanning, and wood preserving. Exposure to chromium can occur through inhalation, ingestion in drinking water and food, or through skin contact with chromium or chromium-containing compounds. The health effects associated with exposure to chromium III and chromium VI are discussed separately below.

CHROMIUM III

At low levels, chromium III is an essential nutrient required for normal energy metabolism. At higher levels, exposure to chromium III has been associated with allergic responses including asthma and dermatitis in sensitized individuals. Occupational inhalation exposure has been associated with gastrointestinal effects including stomach pains, cramps, and ulcers.

CHROMIUM VI

In general, chromium VI is more toxic to humans than chromium III. Exposure to chromium VI has been associated with the adverse health effects mentioned above for chromium III as well as those listed below.

Occupational inhalation exposure to relatively high levels of chromium VI has been associated with dizziness, headaches, weakness, respiratory tract irritation, gastrointestinal effects, liver and kidney effects, and increased risk of death from lung cancer. Ingestion of high levels of chromium VI has been linked to adverse effects on the respiratory tract, cardiovascular system, gastrointestinal tract, white blood cells, kidneys, liver, and nervous system. Adverse health effects associated with ingestion may lead to death. Dermal exposure of workers to chromium VI has been shown to cause deeply penetrating skin ulcers and systemic effects including effects on the kidneys, blood cells, cardiovascular system, and the gastrointestinal tract. The systemic effects seen following dermal exposure can lead to death. An increased incidence of lung cancer in humans has been associated with exposure to chromium VI. There is no evidence indicating the carcinogenicity of chromium III in humans or animals. The EPA has categorized chromium VI in Group A, human carcinogen.

References

ATSDR (Agency for Toxic Substances and Disease Registry). 1993. Toxicological Profile for Chromium. U.S. Department of Health and Human Services. Atlanta, GA. PB93-182434.

EHRAV (Electronic Handbook of Risk Assessment Values). 1994. Electronic Handbook Publishers. November 1994.

HEXACHLOROBENZENE

Hexachlorobenzene (HCB) is formed as a waste product in the production of several chlorinated hydrocarbons and is a contaminant in some pesticides. HCB is a very persistent environmental chemical due to its chemical stability and resistance to biodegradation. Humans can be exposed to HCB in a variety of ways. Occupational exposure can occur at chemical plants manufacturing chlorinated solvents and some pesticides. Farmers can be exposed in applying pesticides in which HCB is a contaminant or by handling contaminated soil. General population exposure will occur in people living near these industrial plants and through consumption of contaminated drinking water. However, the majority of general population exposure will occur through the consumption of food and milk, both of which frequently have detectable levels of HCB.

Effects from acute exposure to HCB in humans may include eye, dermal and mucous membrane irritation, CNS excitation, seizures, pneumonitis, and respiratory depression. Chronic exposure has been reported to cause porphiria cutaneatarda. This condition can cause rash, changes in skin pigment, skin thickening, red or dark urine and other body changes. High or repeated exposure may damage the liver, immune system, thyroid, kidneys and nervous system. Irritability, muscle weakness, tremors, pins and needles, and other nerve damage can occur.

The toxicity of long-term dietary exposure of humans to HCB was demonstrated by the epidemic of porphyria cutanea (PCT) in Turkish citizens who accidentally consumed bread made from grain treated with HCB. In children less than 1 year of age, pink sore disease was observed with 95% mortality. In addition to the PCT-associated symptoms, the exposure caused neurotoxicity and liver damage. Follow-up studies twenty years after the incident in Turkey, some of the individuals were still uffering from the effects of HCB. These studies reported PCT symptoms, reduced growth and arithritic changes in the appendages of children who were directly or indirectly (i.e., through breast milk) exposed.

HCB, when administered orally, has been shown to induce tumors in the liver, thyroid and kidney in three rodent species. Based on these studies HCB has been classified by the EPA as a Group B2, probable human cacinogen.

References

HSDB (Hazardous Substance Data Bank). 1994. National Library of Medicine. Bethesda, MD (CD-ROM Version). Micromedex, Inc. Englewood, CO (Edition expires 1/31/95).

IRIS (Integrated Risk Information System). 1994. U.S. Environmental Protection Agency. Washington, DC (CD-ROM Version). Micromedex, Inc. Englewood, CO (Edition expires 1/31/95).

Children are more susceptible to lead toxicity than adults because they engage in physical activities associated with significant hand-to-mouth ingestion of nonfood items containing lead, and are physiologically more prone than adults to develop high circulating blood levels following exposure. Bone acts as a repository for ingested and inhaled lead. and may be a source of blood lead during growth, pregnancy, disease, or stress. No systemic toxicity from chronic lead exposure is estimated to occur if the lifetime daily intake is less than 0.3 mg.

Genotoxic and cancer-causing effects of lead in human and animal studies are the subject of current debate. The only consistent reports of lead genotoxicity comes from plant studies. Reports of kidney tumors have been reported in animals and humans; however, human tumor incidence is not statistically significant. Environmental lead exposures and potentially cancer-causing doses of lead have been difficult to measure in human epidemiological studies. Based on sufficient animal evidence, EPA has categorized lead as a Group B2, probable human, carcinogen.

The most sensitive target in children for the adverse noncancer effects of lead is the nervous system. For adults, it is the blood and heart. EPA and the Centers for Disease Control (CDC) have developed a range of blood lead levels that may be associated with specific toxic effects. Toxicity can range from subtle neurobehavioral effects in children (e.g., decreased learning performance, small deficits in intelligence scores; 10-15 μg/dL) to severe brain damage in adults or children (80-100 μg/dL). Both prenatal and postnatal lead exposure are influential on postnatal neurobehavioral performance. The critical toxic effect in middle-aged adult males is high blood pressure (5 to 30 μg/dL). Lead may also affect a variety of other organs at intermediate blood levels. Kidney damage, anemia (similar to that caused by iron deficiency), muscle paralysis, and severe vomiting and stomach pain may occur. Effects on the human immune system are inconsistent; positive results tend to be at high blood lead levels. Encephalopathy (brain swelling) is the most life-threatening effect of lead toxicity, and typically occurs at blood levels of 80 μg/dL or higher. Severe lead toxicity may cause sterility, abortion, and infant mortality.

Reference

ATSDR (Agency for Toxic Substances and Disease Registry). 1993. Toxicological Profile for Lead. U.S. Department of Health and Human Services. Atlanta, GA. PB93-182475.

AR308585

MERCURY

Mercury is highly toxic to both humans and animals, the primary targets of toxicity being the kidneys and the nervous system. When ingested, organic mercury is more toxic than inorganic mercury salts and elemental mercury. Data regarding the toxic effects of mercury through inhalation are limited mainly to exposure to elemental mercury vapor. Dermal exposure to mercury can also cause systemic toxic effects.

Considerable evidence indicates that the toxic effects of mercury on the kidneys are due to an autoimmune response. In laboratory animals, the susceptibility to mercury-induced autoimmune kidney damage appears to have a genetic basis.

Both the central and peripheral nervous systems are major sites of mercury toxicity. Exposure to elemental mercury vapor primarily affects the central nervous system, resulting in symptoms that include tremors, insomnia, nervousness, short-term memory loss, decrease in psychomotor skills, and slowed nerve conduction. There are limited data regarding the neurological effects of exposure to inorganic mercury; a few cases of dementia have been reported. Organic mercury, particularly methylmercury, is a potent neurotoxin and is associated with numerous symptoms, including a tingling sensation in the extremities, multiple sensory impairment, loss of coordination, muscle weakness, speech impairment, and depression. The developing nervous system is especially sensitive to organic mercury poisoning; prenatal exposure can lead to severe brain damage and mental retardation.

There are data that suggest that mercury may be genotoxic. There are no studies that indicate that mercury is carcinogenic; however, methylmercury has been shown to increase the formation of urethane-induced lung tumors in mice.

Reference

ATSDR (Agency for Toxic Substances and Disease Registry). 1992. Toxicological Profile for Mercury. U.S. Department of Health and Human Services. Atlanta, GA. PB90-181256.

beta-Naphthalamine was released into wastewater principally from the dye and pigment industry where it is used as a chemical intermediate, however as of 1976 it was no longer produced or consumed commercially in the U.S. because of its ability to cause cancer. Cigarette smoke contains beta-Naphthylamine and may be its most ubiquitous source. Humans can also be occupationally exposed to beta-Naphthylamine from industries where nitrogen-containing organic matter is pyrolyzed.

beta-Naphthylamine is toxic by ingestion, skin absorption, inhalation, and skin or eye contact. Individuals with reduced immunological competence, those undergoing treatment with steroids or cytotoxic agents, pregnant women, and cigarette smokers are at special risk to the health effects caused by beta-naphthylamine. The target organs of beta-naphthylamine exposure are the bladder and skin. Symptoms from acute poisoning may include dermatitis, hemorrhagic cystitis, dyspnea, ataxia, methemoglobinemia, hematuria and dysuria. Some related chemicals can damage the liver and/or cause skin allergy. It is not known whether beta-naphthylamine also has these effects.

beta-Naphthylamine is believed to be one of the most potent industrial carcinogens ever encountered. This chemical has a latent period of approximately 16 years. NIOSH has stated beta-naphthylamine's target cancer site is the bladder. IARC classifies beta-naphthylamine as a Group 1, human carcinogen. beta-Naphthylamine has not been quantitatively evaluated by EPA.

Reference

HSDB (Hazardous Substance Data Bank). 1994. National Library of Medicine. Bethesda, MD (CD-ROM Version). Micromedex, Inc. Englewood, CO (Edition expires 1/31/95).

New Jersey Hazardous Substance Fact Sheets, Right to Know Program. 1994. New Jersey Department of Health. Trenton, NJ (CD-ROM Version). Micromedex, Inc. Denver, CO (Edition expires 1/31/95).

NIOSH Pocket Guide to Hazardous Chemicals. June 1994. U.S. Department of Health and Human Services.

AR308587

NICKEL

Soluble nickel compounds are considered the most toxic of all the forms of nickel. Nickel causes adverse effects in humans at elevated concentrations through inhalation, oral, and dermal exposure. The respiratory system is the main organ of concern following the inhalation of nickel. Limited information shows that the oral route affects the gastrointestinal, hematological, and cardiovascular systems. Occupational nickel and environmental nickel exposures mainly cause allergic contact dermatitis.

In somes studies of nickel-exposed workers, deaths have been associated with benign respiratory disease, and nasal and lung cancers. However, other studies did not indicate an increase in deaths as a result of respiratory disease. Studies in workers exposed to nickel showed no increase in deaths due to cardiovascular disease. Both human and animal studies show that deaths are not likely to occur due to exposure to nickel at concentrations normally present in the environment or at hazardous waste sites. Some of the respiratory effects found in workers following the long-term inhalation of nickel dust are chronic bronchitis, emphysema, and reduced vital capacity. These data were not conclusive due to the fact that the workers were also exposed to other toxic metals. Workers who drank elevated nickel-contaminated water from a fountain experienced nausea, cramps, diarrhea, and vomiting; muscular pain was reported in one worker; a short-term increase in blood reticulocytes, serum bilirubin levels, and urine albumin was also noted. Inhalation exposure to nickel has not caused gastrointestinal effects in either humans or animals.

The predominant nickel effect in the general population is contact dermatitis, which usually results from long-term exposure of the skin to metallic nickel. Human studies indicate that neurological effects such as giddiness, weariness, and headache might occur from short-term or long-term exposures to nickel. Although nickel causes developmental and reproductive toxicity in animals, it is not known if it could cause the same effects in humans.

Limited human studies showed that nickel produces genotoxic effects. Nickel is an occupational cancer-causing agent. Nickel refinery dust and nickel subsulfide are designated as Class A, human carcinogens, by the EPA.

Reference

ATSDR (Agency for Toxic Substances and Disease Registry). 1993. Toxicicological Profile for Nickel. U.S. Department of Health and Human Services. Atlanta, GA. PB93-182491.

AR308588

2,3,7,8-TETRACHLORODIBENZO-p-DIOXIN (2,3,7,8-TCDD)

2,3,7,8-TCDD is neither naturally occuring nor manufactured directly in industry. Rather, it is an impurity from the manufacture of herbicides and germicides, and from the incineration of municipal and industrial wastes. People may be exposed to 2,3,7,8-TCDD by inhaling contaminated air, consuming contaminated food and milk, or directly contacting contaminated soil, vegetation, or industrial byproducts.

No studies were located regarding the inhalation of 2,3,7,8-TCDD by humans or animals. Toxic effects such as chloracne, immunotoxicity, hyperpigmentation, hyperkeratosis, hirsutism, hepatotoxicity, hypertriglyceridemia and hypercholesterolemia, aching muscles, weight loss, gastrointestinal disorders, and neurological disorders have all been observed in humans following ingestion and dermal exposure to chemicals containing 2,3,7,8-TCDD (dose and duration were not reported). However, it is not clear whether these results were due to exposure to 2,3,7,8-TCDD or to the chemicals/solvents contaminated with 2,3,7,8-TCDD.

Studies on animals have concluded that acute and subchronic oral and dermal exposure can yield toxic effects on development, reproduction, the skin, and the liver. Wasting syndrome, liver toxicity, immunotoxicity, and decreased longevity have occurred in guinea pigs following subchronic oral exposure to 2,3,7,8-TCDD. Subchronic dermal exposure of mice has generated dermal lesions. In rats, chronic oral exposure has resulted in toxic hepatitis and degenerative hepatic changes, dilated renal pelvises, gastrointestinal disorders, and decreased fetal weight. Chronic oral studies with rats and mice have revealed an increase in tumors in the lungs, liver, hard palate, and nasal turbinate.

EPA has classified 2,3,7,8-TCDD in Group B2 when the chemical is evaluated alone, and in Group B1 when it is assessed in combination with phenoxyherbicides and/or chlorophenols. Group B2 indicates that there are sufficient animal data to suggest that 2,3,7,8-TCDD is a probable human carcinogen. Group B1 denotes that adequate animal data and limited human data suggest 2,3,7,8-TCDD is a probable human carcinogen.

References

ATSDR (Agency for Toxic Substances and Disease Registry). 1989. Toxicological Profile for 2,3,7,8-TCDD. U.S. Department of Health and Human Services. Atlanta, GA. PB89-214522.

U.S. EPA (United States Environmental Protection Agency). 1994. Health Effects Assessment Summary Tables. FY-1994 Annual. Office of Solid Waste and Emergency Response. PB94-921199.

POLYCYCLIC AROMATIC HYDROCARBONS (PAHs)

PAHs can be produced anthropogenically from the burning of coal, oil, gas, garbage, or other organics, as well as naturally from forest fires and volcanic eruptions. They are mainly used for research purposes, although a few of the chemicals are used in dyes, plastics, and pesticides. PAHs are widespread in the environment and are found in air (in vapor and particulate forms), soil, sediment, and water. Most PAHs do not exist alone in the environment, but are found in mixtures of at least two PAH compounds. There are over one hundred individual PAHs. The following chemicals are considered as one group in this profile:

- Acenaphthene
- Acenaphthylene
- Anthracene
- Benzo(a)anthracene
- Benzo(a)pyrene
- Benzo(b)fluoranthene
- Benzo(g,h,i)perylene
- Benzo(k)fluoranthene
- Chrysene
- Dibenzo(a,h)anthracene
- Fluoranthene
- Fluorene
- Ideno(1,2,3-cd)pyrene
- Phenanthrene
- Pyrene

These compounds usually occur together in the environment and may have similar toxicological effects. People may be exposed to PAHs in the home, workplace, and environment. Nonoccupational PAH exposure occurs through the inhalation of tobacco smoke and smoke from burning wood, and from the ingestion of contaminated water, smoked meats, contaminated grains and vegetables, and processed foods. The greatest potential exposure for most people results from either working or living in areas surrounding coal-tar production plants, coking plants, asphalt production facilities, smoke houses, power and heat generating stations, coal-tarring activities, and municipal trash incinerators.

Studies regarding human exposure to PAHs are limited; most of the information is provided from occupationally-exposed coal and coke workers. Coal tar and its byproducts have been associated with bronchogenic cancer, buccal-pharyngeal cancer, cancer of the lip, gastrointestinal cancers, bladder cancer, scrotal cancer, and skin tumors. However, cancer induction by PAHs and other chemicals may have a synergistic relationship, implying that the carcinogenic qualities of PAHs may be augmented when present with other industrial byproducts. Other studies have revealed that chronic exposure may also have noncancer effects including ocular photosensitivity and irritation, respiratory irritation, cough, bronchitis, dermatitis and hyperkeratosis, and leukoplakia. One study reported an increased incidence of melanosis of the colon and the rectum following chronic ingestion of anthracene-containing laxatives. Tissues with rapid cellular regeneration such as bone marrow, intestinal epithelium, lymphoid tissues, and some reproductive tissues may be more susceptible to PAH toxicity.

Certain subsections of the population may be more susceptible to PAH toxicity than others. These subsections include people with genetically inducible aryl hydrocarbon hydroxylase (AHH) activity, nutritional deficiencies, genetic disease that influence the efficiency of DNA

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repair, immunodeficiency due to age or illness, and fetuses. Other susceptible populations to PAH toxicity include smokers, people who have experienced excessive sun exposure, people with liver or skin diseases, and women, especially of child-bearing age.

The acute ingestion of anthracene, benzo(a)anthracene, benzo(a)pyrene, and phenanthrene has generated enzyme alterations in animal gastrointestinal mucosa. Lethal hematopoietic effects, including aplastic anemia and pancytopenia, have been reported in mice following the acute ingestion of benzo(a)pyrene. Hepatotoxicity studies in animals have shown that the acute ingestion of benzo(a)pyrene induces preneoplastic hepatocytes, which have been correlated with the development of cancer. The acute ingestion of benzo(a)pyrene and benzo(a)anthracene has also increased liver weight and altered liver enzyme production in rats. Liver regeneration, following acute oral exposure to PAHs, increased in studies performed with rats. The acute ingestion of benzo(a)pyrene by pregnant rats and mice has been shown to decrease pup weight and increase the incidence of sterility in F1 progeny. Adverse dermatological effects, including the destruction of sebaceous glands, skin ulcerations, hyperplasia, and hyperkeratosis, have been documented in animals following acute and subchronic dermal exposure.

Hematological effects (e.g., aplastic anemia and pancytopenia) have been observed in mice following subchronic oral exposure to benzo(a)pyrene. Similarly, rats chronically fed PAHs have developed agranulocytosis, anemia, leukopenia, and pancytopenia. Decreased kidney size, congestion, and renal cortical hemorrhages have been observed in rats exposed to various PAHs.

Skin, lung, liver, and gastric cancer have all been produced in laboratory animals chronically exposed to various PAHs. Benzo(a)anthracene, benzo(a)pyrene, and dibenzo(a,h)anthracene have also been found to be carcinogenic when administered orally to animals. Benzo(a)pyrene also has been found to be carcinogenic when applied dermally to animals. PAHs extracted from coal furnaces have caused skin tumors in mice following chronic dermal administration. Data have suggested that skin tumors are primarily due to benzo(a)pyrene, alone or in combination with dibenzo(a,h)anthracene. However, studies have also found benzo(a)anthracene, chrysene, benzo(b)fluoranthene, and benzo(k)fluoranthene to induce skin tumors in mice and rats.

The most significant PAH toxicity endpoint is carcinogenicity. Based on available human and animal evidence, EPA has classified individual PAHs as follows:

- Group B2 (probable human carcinogen): benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, chrysene, dibenzo(a,h)anthracene, and ideno(1,2,3-cd)pyrene.

- Group D (not classifiable as to human carcinogenicity): acenaphthylene, anthracene, benzo(g,h,i)perylene, fluoranthene, fluorene, phenanthrene, and pyrene.

- The carcinogenic classification for acenaphthene is pending.

References

ATSDR (Agency for Toxic Substances and Disease Registry). 1990. Toxicological Profile for Benzo(a)anthracene. U.S. Department of Health and Human Services. Atlanta, GA. PB90-247669.

ATSDR (Agency for Toxic Substances and Disease Registry). 1990. Toxicological Profile for Benzo(a)pyrene. U.S. Department of Health and Human Services. Atlanta, GA. PB90-258245.

ATSDR (Agency for Toxic Substances and Disease Registry). 1990. Toxicological Profile for Benzo(b)fluoranthene. U.S. Department of Health and Human Services. Atlanta, GA. PB90-247651.

ATSDR (Agency for Toxic Substances and Disease Registry). 1990. Toxicological Profile for Chrysene. U.S. Department of Health and Human Services. Atlanta, GA. PB90-247644.

ATSDR (Agency for Toxic Substances and Disease Registry). 1990. Toxicological Profile for Polycyclic Aromatic Hydrocarbons. U.S. Department of Health and Human Services. Atlanta, GA. PB91-181537.

TOMES(R) Information System. 1994. Hall, A.H. and B.H. Rumack (eds.). Micromedex, Inc. Denver, CO (Edition expires 7/31/94).